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SATURDAY JUNE 28 1952

TABLE OF CONTENTS

Infective Hepatitis: A Problem of World Health SIR JOHN MCNEE, M.D., F.R.C.P. 1367
Structural and Functional Adaptation in Renal Failure.—II ROBERT PLATT, M.D., F.R.C.P. 1372
Sympathectomy for Raynaud's Disease
J. B. KINMONTH, M.S., F.R.C.S., and G. J. HADFIELD, F.R.C.S. 1377
Septic Jaundice D. B. IRWIN, M.D., M.R.C.P. 1379
Vitamin K in Treatment of Acute Hepatitis HANS-OLOF MOSSBERG, M.D. 1382
Stress Incontinence G. GORDON LENNON, CH.M., F.R.C.O.G. 1384
Icterus Gravis and Cirrhosis of the Liver JOHN GERRARD, D.M., M.R.C.P. 1385
Effect of Liver Supplement on Children's Growth JOHN YUDKIN, M.D., M.R.C.P. 1388
Tuberculosis Survey in Schoolchildren in the Calder Valley
S. KEIDAN, M.B., C.P.H., J. LYONS, M.B., D.P.H., BERTRAM MANN, M.D., D.P.H., and G. A. WILTHEW, M.B., B.S. 1390

MEDICAL MEMORANDA

A Case of Prostatic Abscess. ERIC DUNLOP, M.D., M.R.C.P., and JOHN HOSFORD, M.S., F.R.C.S. 1392
Testing for Serratus Anterior Weakness. CYNTHIA M. WALKER 1393

REFRESHER COURSE

Clinical Effects of Exposure to Heat and Sunlight—II. B. G. MAEGRAITH, D.PHIL., M.B., M.R.C.P. 1402

GENERAL ARTICLES AND NEWS

NOVA ET VETERA 1404
MEDICAL NOTES IN PARLIAMENT 1412
UNIVERSITIES AND COLLEGES 1413
MEDICAL NEWS 1415
BIRTHS, MARRIAGES, AND DEATHS 1416
QUESTIONS AND COMMENTS 1417

REVIEWS

Surgery of the Brain 1394
Photography of Patients 1394
Social Medicine 1394
Asthma 1394
Blood Groups and the Law 1395
Malaria 1395
Dublin—A City with a Tradition 1395

MEDICO-LEGAL

Doctor's Action against Ex-partners Fails 1412

LEADING ARTICLES

Renal Failure 1396
Tuberculous Infection 1397
Epidemiology of Virus Hepatitis 1399

ANNOTATIONS

Organization for Medical Mycology ..1400
Intravenous Feeding with Fat Emulsions 1400
Vitamin B_{12} and Growth 1401

OBITUARY

J. F. Taylor, M.D. 1410
H. W. Grattan, C.B.E., D.S.O., M.R.C.S. ..1411
W. S. V. Stock, F.R.C.S. 1411
William Ibbotson, F.R.C.S.Ed. 1411
A. M. Amsler, M.D. 1412

CORRESPONDENCE

W.H.O. and Birth Control. DAVID PYKE, M.B. 1405
Trichlorethylene. GORDON OSTLERE, M.B. 1405
Antihistamines and Skin Eruptions. G. W. LEWIS, M.B. 1405

SUPPLEMENT

Full Contents on First Page of Supplement

Joint Annual Meeting—Dublin, July 3–11, 1952 329
A College of General Practice 335
Some Things the B.M.A. Did in 1951 ..340
General Medical Services Committee ..342

CORRESPONDENCE—*cont.*

Pulmonary Embolism. H. M. HANSCHELL, M.R.C.S.; A. J. PARTRIDGE, F.R.C.S. ..1405
Mepacrine. W. G. TILLMAN, M.D. 1406
Metallic Mercury. D. H. JUDSON, M.B. 1406
Dermatologist's Protest. F. F. HELLIER, M.D. 1406
Responsibility and Registration. P. H. SHERWOOD and D. G. SHERWOOD, M.B. 1406
Erythrocyte Sedimentation in Anaemia. RICHARD TERRY, M.D. 1407
Liaison Needed. G. M. WODDIS, M.R.C.S. 1407
Skin Lesions in Ulcerative Colitis. C. L. HEANLEY, F.R.C.S. 1407
Torsion of Fallopian Tube. W. G. MILLS, F.R.C.S. 1407
Wandering Spleen. J. M. WARD, M.R.C.S. 1407
Bladder Paralysis. H. R. ALDERTON, M.B. 1408
Fractured Clavicle. L. J. LEVENE, M.B. 1408
Migraine. L. J. MOIR, M.B. 1408
Growth of the Foetal Head. H. CAMPBELL, B.A., and others 1408
Vascular Changes During Anaesthesia. K. E. COOPER, M.B., and others 1409
Cancer. GUY BOUSFIELD, M.D. 1409
Employment for Over-sixty-fives. J. ARMSTRONG, F.R.C.S.I. 1409
Myositis Ossificans. R. H. MAUDSLEY, F.R.C.S. 1410
College of General Practice. J. H. HUNT, D.M. 1410

Vital Statistics 1414

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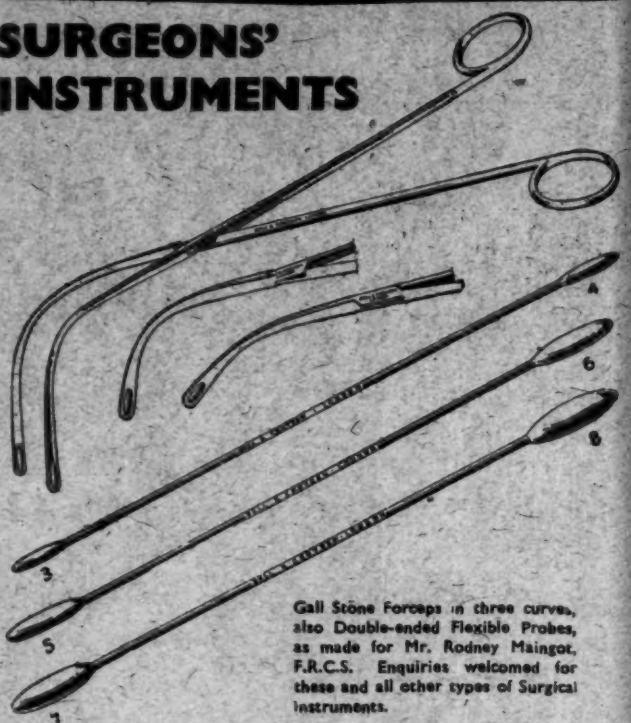

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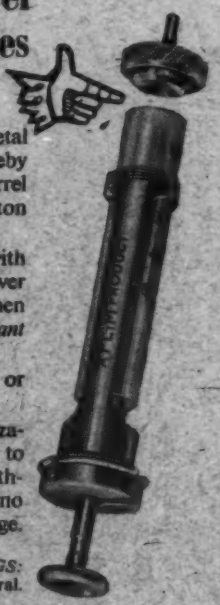

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY JUNE 28 1952

INFECTIVE HEPATITIS: A PROBLEM OF WORLD HEALTH *

BY

Sir JOHN McNEE, M.D., D.Sc., F.R.C.P.

Regius Professor of Practice of Medicine, University of Glasgow

I have chosen infective hepatitis as the subject of my Harveian Lecture for several reasons. First of all, I have been interested throughout practically the whole of my professional life in the problems of hepatic disease and of jaundice, and have taken some small part in their investigation in Britain during the past 30 years. Next, the main disease which I propose to discuss—under a different name from that which I was first taught to employ—has not only an interesting ancient history but a quite astonishing modern one; and has proved itself to be, from numbers alone, the most important of all the diseases of the liver associated with jaundice. Lastly, although scarcely on the same scale as malaria and influenza, this disease has shown itself capable in favourable circumstances of spreading widely in pandemic form, and might easily do so again.

I later refer to two other forms of infective hepatitis, either very closely allied to or, according to one view, identical with acute infective hepatitis of the common kind—namely, homologous serum jaundice and syringe-transmitted hepatitis. Neither of these was known to exist until comparatively recent times.

This disease-group, if I may call it so at present, has been clearly proved to result from virus infection, and our difficulties in its investigation simply emphasize that we are still at an early stage in our knowledge of all virus diseases and their propagation.

The History of Catarrhal Jaundice

Catarrhal jaundice was the name used by clinicians for many years to describe the disease now more accurately classified as acute infective or viral hepatitis. It was well known first only in its *sporadic form*, but even then was recognized as by far the commonest type of jaundice seen in clinical practice in Britain, Europe, and America. Its occurrence in epidemic form, both in peace and in war, is described later. Sporadic catarrhal jaundice was always recognized as benign, and the rare progression to acute yellow atrophy (acute necrosis) of the liver was taken to be the result of a different disease. Its onset was always associated with gastro-intestinal upset, and when Virchow (1864) described the pathology in a single case as a catarrhal obstruction of the common bile duct, the mechanism of the disease appeared to be reasonably explained and its first name justified. It was assumed that a microbic infection, not necessarily specific, spread upwards from the disturbed intestine to block the bile duct by catarrhal inflammation or cholangitis. This fitted the accredited view in the early years of this century that, as Eppinger (1908) taught, all varieties of jaundice were essentially obstructive, whether the obstruction occurred in the larger extrahepatic ducts (as in catarrhal jaundice) or in the finest bile capillaries within the liver (as in cirrhosis).

This conception of the pathology of catarrhal jaundice, although doubted by a few, continued until after the end of the first world war, when certain new discoveries about hepatic diseases and about the mechanics of jaundice were made, and could not be denied. The old conception, however, died hard; and I recall with some amusement the many friendly arguments I had with the late Sir Arthur Hurst until he finally recanted about the disease we are now discussing.

I should like to add here, in fairness, that I have never denied the possibility of an ascending cholangitis spreading up from the intestine to produce a true catarrhal and obstructive jaundice. I have notes of cases of this kind which exhibit a striking and unusual clinical history, and some day I hope to write a full account of them.

The new developments in the early years after the first world war cannot easily be placed in chronological order, so I shall place them in order of importance. The first, in my own view, was the discovery by Hijmans van den Bergh, of Utrecht (actually in 1913, but known in Britain only in 1918), of his method of estimating small amounts of bile pigment in small amounts of blood serum. This method, now so well known as the van den Bergh reaction, brought about a revolution in our whole knowledge of the production of jaundice. It gave us the modern definition of jaundice as essentially "bilirubinaemia," and when exploited as a clinical and experimental means of investigation it made possible a new and rational classification of jaundice (McNee, 1922–3) into three varieties—obstructive, toxic and infective (hepatitic), and haemolytic—which is still in common use to-day.

The other new development was the gradual realization of the conception of "hepatitis"—the idea that by far the majority of diseases or disorders of the liver are essentially due to damage, sometimes temporary but often permanent, leading to death of the glandular cells of the organ. This conception grew so slowly that its origins cannot accurately be traced, but the van den Bergh technique added greatly to its acceptance and proof.

*The Harveian Lecture (1952) of the Harveian Society of London, delivered on March 20 at the Royal College of Surgeons of England.

My own first experience of a genuine hepatitis came during the first world war when Weil's disease (spirochaetosis icterohaemorrhagica) broke out in Flanders (McNee, 1920). The spirochaetal cause had just been discovered, fatal human cases were examined *post mortem*, and, best of all, the whole evolution of the damage to the liver cells and the processes of repair could be studied in guinea-pigs inoculated with the disease.

With this and other experiences in mind, some dating from before the war and concerned with epidemics, the probability that the old catarrhal jaundice was pathologically a hepatitis—generally benign but sometimes going on to acute necrosis (yellow atrophy)—was strongly suggested. Final proof, in Britain at least, was long in coming, for patients do not die in the earliest stages of the disease. Opportunities came, however, first to Gaskell (1933) in Cambridge, and later to Barber and Osborn (1939) in Derby, of examining the tissues of two patients who died after an operation and as the result of an accident, and in both cases a typical hepatitis was found. In Rolleston and McNee's textbook (1929) the disease was described as common infective hepatic jaundice, but this has gradually been superseded by acute infective hepatitis.

Epidemics of Jaundice in War and Peace

Simply for convenience, epidemics of jaundice, clearly due as we now know to different causes, may be divided into those occurring in wars and those occurring in countries or communities in peace. War epidemics have always been the greater, and are considered first; but in modern "total war" civilians may suffer almost as much as the fighting forces.

The growth of bacteriology has enabled us to separate clearly some of the clinical varieties of jaundice, but even then several recognizable causes may be present at the same time and lead to diagnostic difficulties. What we now know as Weil's disease, a rat-borne infection due to a spirochaete, was evidently quite common in many campaigns, especially when, as in the good old days, the belligerents and their horses retired to winter quarters. Malaria, bacillary dysentery, and the enteric group of fevers, all potential causes of jaundice in a proportion of sufferers, have often occurred together in war and led to confusion.

The real question before us is whether what was often loosely termed "campaign jaundice" included many cases of acute infective hepatitis. The largest outbreak of all, referred to both by Willcox (1916) and by Von Bormann *et al.* (1943), seems to have been during the American Civil War, when 22,509 cases were reported, with 161 deaths. This was before the advent of bacteriology, but the benign nature of the disease must leave us with a strong suspicion of infective hepatitis. In the South African War 5,648 cases of jaundice are recorded with a small mortality and little bacteriology.

In the first world war, to my own personal knowledge, epidemics of jaundice, except for the small one soon identified as spirochaetosis icterohaemorrhagica, were never noted among the millions of British and Allied troops deployed in the trenches of Flanders. In the Eastern Campaign, particularly in the Dardanelles and in Mesopotamia, the circumstances were quite different, and have been fully described by Willcox. No actual figures for the Dardanelles campaign are available, but careful examination on the spot by bacteriologists of repute, such as C. J. Martin and Ledingham, failed in by far the majority of cases to reveal a bacterial cause such as typhoid, paratyphoid, or dysentery. Virus investigation was then scarcely in its infancy, but it now seems reasonable to conclude that most of the many jaundiced men suffered from acute infective hepatitis. In Mesopotamia in 1917–18, among a much smaller number of troops, it is known (Willcox) that 2,403 cases of jaundice occurred in British soldiers with 18 deaths, and 3,897 in Indian soldiers with 28 deaths.

In countries and communities at peace the history of epidemics of jaundice is a long one, but as none of the outbreaks until recent times was large they failed to attract more than local attention. The best historical account I have read is by Von Bormann *et al.* (1943), stimulated by their interesting and careful investigation of a number of small epidemics in German villages in 1937–8. By this time they were firmly able to give as the title of their monograph, "Hepatitis Epidemica in Deutschland." Their historical researches take them back as far as 1629, and they refer to an outbreak in the English Army in Flanders in 1743. They also state that the first definite description of a civilian epidemic labelled "icterus epidemicus" was given by Herlitz in Göttingen in 1791.

For reasons already described, however, and with the great advances in our knowledge in the early nineteen-twenties, it is only then that civilian outbreaks of jaundice began strongly to attract attention and rigid investigation of their cause. In Britain credit for attracting attention to outbreaks in children and the obvious infective character of the disease is due to Cockayne (1912). Between the wars many small epidemics were reported, chiefly affecting schoolchildren, and particular attention must be drawn to the work of Pickles (1939) in a country practice, and to the paper by Cullinan (1939). I took part in several investigations myself, in association with my colleague Okell at University College Hospital, but our results were negative, and we knew nothing then about virus infections. All we recognized was the obvious infectivity—over 60% of the pupils in one preparatory school—and that the probable incubation period was round about 30 days. It became clear from the many small outbreaks in different parts of Britain that by 1939 the infecting agent was widely spread throughout both country districts and the towns.

At the same time small epidemics were occurring in many European countries—in Germany, and particularly in Scandinavia, where severe and fatal infections took place in adults as well as children.

Relations of Homologous Serum Jaundice and Syringe-transmitted Jaundice to Acute Infective Hepatitis

It is true that the term "homologous serum jaundice" came into use in Britain only after publication of the memorandum prepared by medical officers of the Ministry of Health (1943). I have always understood that Dr. W. H. Bradley had a large share in the preparation of this invaluable document. It seems almost certain that syringe-transmitted hepatitis is the same disease, and I wish to recall that syringe-transmitted jaundice has an older history, not mentioned in the above memorandum, and tending to be forgotten. Omitting subcutaneous injection of morphine and a few other drugs, large syringes and long needles came into common use only with the advent of "salvarsan" about 1909. I can remember learning the technique with poor syringes until I obtained a German Luer type, and I can also remember the services of a surgeon being required for the "dangerous" operation of lumbar puncture. Not long after the extended use of salvarsan compounds intravenously, concern began to be expressed over sudden outbreaks of jaundice in V.D. clinics. The drug was naturally suspect, as a potential hepatic poison; and this led to the setting up of a Salvarsan Committee of the Medical Research Council, which published two reports, in 1919 and after the first world war in 1922. I shall make reference only to the second report, since I was then a member of the committee and knew the facts now to be described at first hand.

In 1917 an outbreak of jaundice, with nine deaths, occurred at a barracks in Dublin where V.D. patients were being treated. The injection technique would now be

described as defective, and to make the story short it was proved that the deaths all resulted from the transmission of malignant malaria by injection. This outbreak thus does not directly concern jaundice, but may be one of the first accounts of a fatal disease accidentally transmitted by intravenous injection. In the same year a serious outbreak of jaundice, with 15 deaths, occurred at the V.D. department of Cherryhinton Military Hospital, Cambridge. The Salvarsan Committee could come to no decision on this outbreak, but excluded specially toxic batches of the drug. We can see this outbreak now as one of syringe-transmitted jaundice, and it is of some interest that the committee drew attention to the fact that at the same time there was a small epidemic of jaundice among children in an elementary school near by, affecting 15 children and one adult.

The next outbreaks of what we now term homologous serum jaundice, and the first to attract widespread attention, are well described in the Ministry of Health memorandum, and need only brief reference here. They are concerned with 41 cases of jaundice and eight deaths following injection subcutaneously of measles convalescent serum into children in the South of England in 1937. Two years later a similar incident from a single batch of pooled convalescent serum occurred in Leeds, fortunately without any deaths.

Here indeed was a clinical puzzle which at the time none of us who were consulted could resolve. Moreover, it was noted with astonishment, just as in the "late jaundice" referred to in the M.R.C. report of 1922, that the jaundice did not begin until on an average about three months after the injection. This was an added puzzle, which remains with us to-day, and is discussed later.

The jaundice which may occur at an interval of about three months after blood transfusion may also be left for subsequent discussion, since blood transfusions were comparatively infrequent and this hazard was not recognized until the period of the second world war.

I began to carry out blood transfusions in France as early as 1915, in association with my surgeon friend Hamilton Drummond, of Newcastle-upon-Tyne, using the primitive Kimpton tube, a large bomb-like container of glass tapering to a glass cannula at one end. Some of our patients were jaundiced when they died, but they were all severely wounded men, mostly with gas-gangrene, and this disease may have accounted for the icterus, although the possibility of incompatibility of the donor's blood cannot be excluded. It seems extraordinary to admit that until Roger Lee, of the Harvard Unit from Boston, first presented us with Type II and Type III sera and taught us to use them to establish the four Moss groups (a classification now superseded) not one of us in France knew anything whatever about blood groups and blood incompatibilities. (A similar gap in knowledge between America and Britain occurred later about coronary thrombosis and its clinical syndrome, and is still quite incomprehensible.)

Once blood grouping was known, and technical improvements were made in methods, I often carried out in the later stages of the war as many as 20 blood transfusions in a single day after a battle, but never heard of jaundice as a result. It would seem that the infective agent which causes homologous serum jaundice was not widely dispersed among the soldier-donors at that time, and it may be added that few of them could possibly have suffered from acute infective hepatitis during their sojourn in France, where no outbreak ever occurred.

Acute Infective Hepatitis and Homologous Serum Jaundice in the Second World War

All that I have said so far leads up to the climax of the second world war, and no one imagined in advance that these two infections, if indeed there are two, would be of such enormous importance as war diseases, not only in the combatant forces of all nations involved but in the war workers at home. It is common knowledge that modern wars are always a powerful stimulus to medical research, and there began at once intensive investigation into the problems of infective hepatitis.

I do not intend to deal with the clinical aspects of the pandemic, which was at its height between 1942 and 1944, and then gradually declined. Everyone, however, is agreed that the acute stages of infective hepatitis and homologous serum jaundice are clinically identical, and that the only real difference is the length of the incubation period.

I may perhaps mention here an important after-effect of the acute hepatitis, fortunately affecting only a small minority of patients. I refer, of course, to the progression to chronic hepatitis or cirrhosis (Lucké and Mallory, 1946; Rennie, 1951), a sequel which may trouble physicians for years to come, and also pension boards.

I prefer in the space available to consider critically the epidemiology, our newly acquired knowledge of aetiology and transmission, and the problem of whether there is only one, or more than one, disease.

There are many gaps in our understanding of why epidemics and pandemics suddenly break out, and about their periodicity. This applies to some virus diseases, such as influenza, which has been most closely studied, but not of course to smallpox. All I have said earlier goes to prove that the infective agent of acute hepatitis was widely spread throughout the world in the years prior to 1939, and, as was noted for the influenza pandemic of 1918–19, that small outbreaks were common for some time before the great explosion.

It is difficult to trace where the main epidemic really started, and I doubt if the war history, when published, can be very informative. My first information came, not from the Navy, but from the account sent by Cameron, of Edinburgh, from Palestine in October, 1942, but not published until the following July (Cameron, 1943). He notes that the epidemic began in troops in Palestine (342 cases) in 1940, and thereafter rapidly increased. It may well be that the explosion took place in different places almost simultaneously, for soon infective hepatitis was widespread over Europe and North Africa. I have seen figures indicating the extent of the pandemic in British and American soldiers, but none concerning our enemies. It must suffice to say that the numbers were so large as to influence war strategy, and both combatant sides were almost certainly equally afflicted.

Homologous serum jaundice first came into prominence as a war disease among our American allies, and followed extensive preventive inoculation with a yellow fever vaccine, containing human serum, prior to their departure from America on foreign service. It has been shown since then by Havens (1946) that as little as 0.01 ml. of infective serum is enough to cause the disease. During the first six months of 1942, just as the pandemic of infective hepatitis was rapidly reaching its zenith, 28,505 Americans inoculated against yellow fever developed jaundice, and 62 died. Since the incubation period was about three months, a number of these men—especially air crews in training—had reached Northern Ireland and East Scotland before they fell ill. I was thus personally able to observe some of them closely. I saw no clinical difference whatever between what I still describe here as the two diseases, except that after homologous serum jaundice recovery seemed slower, and convalescence was often not complete for three months.

British soldiers and airmen, who were not inoculated in this way, naturally escaped this large outbreak, and the first British foretaste of the problem came from the developments of blood transfusion.

Blood transfusion, and especially the preparation and use in battle areas of pooled and stored human blood products of various kinds, developed enormously during the war, and the impetus has of course carried on into peace with the permanent establishment of our blood transfusion services and blood banks.

There has never been any suggestion, so far as I am aware, that much, if any, of the pool blood products had been derived from donors who had recently suffered from infective hepatitis, but there is still a loophole here. One thought instead of the already known puzzle of syringe-transmitted jaundice and the outbreaks of measles convalescent serum jaundice between the wars; and the memorandum of the Ministry of Health, when published, drew at once the strongest attention to this new problem and the necessity to attempt its solution.

The Infecting Agent, or Agents, of Infective Hepatitis and Homologous Serum Jaundice, and their Mode of Transmission

It was shown by Cameron (1943) in Palestine and by Voegt (1942) in Germany that infective hepatitis could be transmitted from man to man by subcutaneous injection of blood in the early stage of the disease. All ordinary bacteriological investigations had always been negative, and no experimental animal had proved susceptible to the disease. The inference seemed obvious that a virus was concerned, and that nothing short of experiments on human volunteers would suffice to give the required knowledge about the disease and its transmission. Such experiments were carried out, actually during the war years, both in Britain and in America, but in modest numbers, for it had to be remembered that the disease carries a small but quite definite mortality rate. This was quite different from the human transmissions of trench fever with which I was actively concerned in the first world war, for there was no fear of fatalities or permanent sequelae. Transmission experiments in the field were forbidden by the British military authorities, and were therefore undertaken at home under the auspices of a committee set up by the Medical Research Council.

An excellent and concise account of the experiments, including both British and American work, is given in the M.R.C. Report (1951). The difficulties were enhanced by the need to find volunteers in different centres, and a very few experiments were carried out in my own department (Rennie and Fraser, 1946). The work was mainly concerned with the transmission of acute infective hepatitis and to a less extent with homologous serum jaundice. This was inevitable because the main and immediate war problem concerned the first condition, but in a way unfortunate because the second remains with us in civilian life as a continuing trouble.

The easiest way of approach, in discussing the human experiments, is that taken in the M.R.C. Report by MacCallum, Stewart, and Bradley, and to consider the possibility of infective hepatitis being due to virus A, and homologous serum jaundice as due to a different virus, B.

It seems to be proved up to the hilt that virus A, derived from patients with acute infective hepatitis, can be transmitted readily to human volunteers by the faeces taken in the early stages of the disease. These experimental observations, moreover, have had striking clinical proof from the observations of Neefe and Stokes (1945) in America on an epidemic of jaundice in a summer camp in which the water supply became contaminated from the latrines. Virus B, from cases of homologous serum jaundice, has seldom been transmitted except by direct injection of blood or serum.

The sum total of all the experiments, both in Britain and in America, does not in my opinion provide adequate proof of the existence of two separate viruses, even when the small number dealing with the question of cross-immunity (Havens, 1945; Neefe, Stokes, and Gellis, 1945) is taken into account. I need not emphasize, however, the extreme difficulty of all such experimental transmissions in a limited number of volunteers in different places, quite apart from the technical problems of the preparation of suitable inocula in the laboratory.

Space alone prevents me from a careful consideration of the individual experiments, but for those who are interested the results are all available in the M.R.C. Report.

Discussion

Acute infective hepatitis, the old catarrhal jaundice, has a long history, and we can read of its sporadic incidence and of occasional epidemics over a period of many years.

Homologous serum jaundice has only a short history, for even if the outbreaks of syringe-transmitted hepatitis in V.D. clinics are included, that takes us no further back than about 1910 at the earliest. Moreover, since the infective agent, so far as is known, is transmitted only by parenteral injection—therapeutic or accidental—of blood or blood products, the possibility of this being a new or hitherto unrecognized disease would, in my view, require the most convincing proof. Such a disease would, on the face of it, seem to stand a poor chance of long survival without a natural means of spread.

It is worth while to compare and contrast the two conditions in the light of our present incomplete knowledge, and note their similarities and differences. There is complete agreement that their clinical features and pathology (Dible, McMichael, and Sherlock, 1943) are identical, but that their period of incubation is different. The means of transmission of homologous serum jaundice seems to be clear-cut and entirely artificial, unless we have been completely led astray. Acute infective hepatitis, on the other hand, can obviously spread rapidly by natural means, but there is not yet full agreement about the portal of entry. Field observations among our combatant forces during the war pandemic strongly suggested the agency of flies and faeces—that is to say, entry by the mouth into the alimentary tract. In civilian outbreaks, especially among children, the majority verdict has always been in favour of droplet-infection through the nasopharynx. These two views do not seem to be completely incompatible, for by both methods the virus would at some time be in the mouth, and it is not necessary to assume that a primary nasopharyngeal infection must always pass on to the trachea and lungs. On the whole, the most likely portal of entry would seem to be the mouth, since we are dealing with a disease which obviously involves the gastro-intestinal system quite early—the stomach first, soon followed by hepatic enlargement and jaundice. The possibility of faecal infection of the fingers in schoolchildren cannot be ignored, especially when one thinks of the notorious frequency of typhoid fever among the nursing attendants of this disease even in well-known hospitals.

The transmission experiments to human volunteers, already briefly described, present real difficulties, and it is true that the relatively small number so far carried out, both in Britain and in America, have strongly suggested to the investigators that there are two diseases—one due to virus A and the other to virus B.

It may seem bold to doubt this conclusion, but, as I have stated, I hold that the experiments so far reported are still quite inadequate for proof. If the careful account of them in the M.R.C. Report is read, the inherent difficulties will be fully realized, and it will be noted how many of the attempted transmissions were negative in comparison with the positives. The only result which seems certain—and it is of the greatest epidemiological and practical importance—is that the virus of acute infective hepatitis is present in the faeces in the acute stages of the disease.

If I suggest, as I now intend to suggest, that we are dealing with only one disease, and not two, what evidence can I marshal in favour of my hypothesis?

I must assume that the single virus, already well spread through the community, especially in children, for a number of years, can survive in the body of a previous sufferer from the disease for a long but indefinite time. To account for homologous serum jaundice my argument would necessitate the presence, from time to time at least, of the virus free in the blood stream. To account for acute infective hepatitis a long-term carrier state with the virus in the faeces is all that need be considered.

Let us now examine these two possibilities. I admit frankly my difficulties in trying to explain the presence of

the virus, even from time to time, in the blood serum of a healthy blood donor with no previous history of jaundice. It is well known that acute infective hepatitis may occur without any obvious trace of jaundice at all, and in civil practice may very easily be missed. I saw many cases of this kind during the war pandemic, but, being on the alert, had no doubts about the diagnosis. I cannot, however, explain the continued presence of the virus in the blood, because I know so little of how viruses live and how long they survive under differing conditions. I can only think of one analogy, from my own clinical experience, which may or may not be significant. I have seen cases of herpes, particularly herpes labialis, in which the virus seems to live quietly for many years but makes its presence known only when some other infective process occurs in the body.

In connexion with a resting reservoir of the virus of infective hepatitis in the faeces of a previous sufferer—recognized or unrecognized—from the acute disease, I have something to say based on my past experience of the carrier state in a bacterial disease.

In the Ypres salient, in the early part of the first world war, I had the remarkable experience, partly shared with the late Adrian Stokes, of hunting out successfully hundreds of typhoid carriers among Belgian refugees who were infecting our troops in their billets behind the lines. These unfortunate carriers were shown to be carriers only when diarrhoea was deliberately induced, and without the field help of the Friends Ambulance Unit and a convenient drug swallowed on the spot (calomel) the labour would have been in vain. I have long concluded, with good clinical evidence to support me (chronic typhoid cholecystitis and gallstones, outbreaks traced to an unrecognized carrier 40 years after the acute fever), that most patients who have had typhoid fever continue to harbour the specific bacilli in their upper intestine for life. When they are well they are safe, for the bacilli are rapidly outgrown in their slow passage through the colon; but when diarrhoea is present they may be active and dangerous carriers of the disease.

I cannot say if this analogy will hold between a bacillus and a virus, but I suggest that this point may be worth bearing in mind when considering the possibility of chronic faecal carriers of infective hepatitis in outbreaks of the disease. This hypothesis could be tested now only by further transmission experiments to human volunteers, and these are unlikely in Britain. Otherwise we must wait until the virus can be cultivated and identified in a laboratory, or some animal is found to be susceptible to the disease.

I must not avoid the problem of the two incubation periods—about 30 days and about 90 days—and it is true that most of the other virus diseases of which I have clinical experience (smallpox, measles, influenza) have a remarkably constant incubation period, or at least one not varying within wide limits. I can only suggest, for clearly I do not know, that there may be some difference, worth investigating in virus laboratories, between a virus when free in the faeces and when contained or constrained in some way in the blood. Lichtman (1949) puts forward a similar point of view, and suggests that the virus may become attenuated in the blood serum.

Prevention and Control

Acute infective hepatitis in its epidemic form is not at present subject to any possible control, and we must await the cultivation of the virus in the laboratory before any question of active immunization could arise. We are really in the same position about acute poliomyelitis, another virus disease in which the faeces may be a reservoir.

Syringe-transmitted hepatitis and homologous serum jaundice obviously could be controlled if we knew a certain way of destroying the virus in the serum. Considerable steps have already been taken in this direction by means of exposure to ultra-violet light, but the results are still insecure.

All pooled quantities of human sera appear to contain antibodies of some kind to the virus of hepatitis in the globulin fraction, and "immune globulin," in doses of 10 ml., has already been used with some success as a preventive after blood transfusion.

This, however, concerns only individuals, and not the great problem of a major epidemic.

Summary

In this Harveian Lecture I have tried to put before you some of my thoughts on acute infective hepatitis and on the conditions known as homologous serum jaundice and syringe-transmitted hepatitis.

I remain so far unconvinced, for reasons I have given, that we are dealing with more than one disease, due to a single virus and not to two.

There are still many things to explain, which only future advances in our knowledge of viruses can make clear.

The virus of acute infective hepatitis is now widely spread throughout the world. The disease occurs sporadically and in small outbreaks at frequent intervals, and has already once exploded in real pandemic form. In these respects it resembles influenza.

Having once rapidly spread as a pandemic, this course of events might be repeated at any future time. In this sense the disease has thus become a problem of world health.

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C. E. Vulliamy, in his book of essays *Rocking Horse Journey* (1952, p. 94), writes: "Miss Cobbe [Frances Power Cobbe, 1822–1904, philanthropist and religious writer] is probably the only person who has ever suggested that our highly respectable scientific publications, the *Lancet* and the *British Medical Journal*, were the corrupters of youth in public libraries. 'Who would have thought 30 years ago,' she wrote, 'of seeing young men in public reading-rooms snatching at the *Lancet* and the *British Medical Journal* from layers of what ought to be more attractive literature and poring over hideous diagrams and revolting details of disease and monstrosity?'"

STRUCTURAL AND FUNCTIONAL ADAPTATION IN RENAL FAILURE*

BY

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One of my objects in presenting the material I am using for these lectures is to illustrate how the investigation of human disease can contribute to physiological knowledge, just as physiology contributes to medicine, and I shall cite some cases in which present physiological theory seems inadequate to account for the findings of the clinician. I intend now to consider the excretion of certain electrolytes and of water, and then to pass on to the more clinical problems of hypertension and renal anaemia.

Excretion of Electrolytes

1. Sodium and Potassium

Electrolytes such as sodium, potassium, phosphate, and chloride are all valuable to the body, and the function of the kidney with regard to them is normally one of conservation rather than excretion. On an average diet something like 99% of the sodium filtered at the glomerulus is returned by tubular reabsorption to the blood stream. As about 99% of the water filtered is also reabsorbed—the normal urine flow being only a very small proportion of the glomerular filtration rate—this means that the renal tubules are usually reabsorbing in large quantities a fluid which is comparable in composition to physiological saline. When a diet almost free from sodium is administered, such as the rice diet sometimes used in the treatment of hypertension, sodium virtually disappears from the urine. Fig. D shows what happened when I went on the rice diet myself for nine days—an experience into which my colleagues persuaded me, and which I do not recommend to anyone but a renal enthusiast, since all meals consist solely of boiled unsalted rice, fruit, and sugar. You will see that the daily sodium excretion comes down from about 200 to about 4 mEq per day, or from about 12 to 0.2 g. in terms of sodium chloride. We have shown elsewhere that this is clearly a function of the tubules (Black, Platt, and Stanbury, 1950), which reabsorb more sodium in these circumstances, and reabsorb correspondingly less sodium when the opposite experiment is performed and hypertonic saline is administered intravenously.

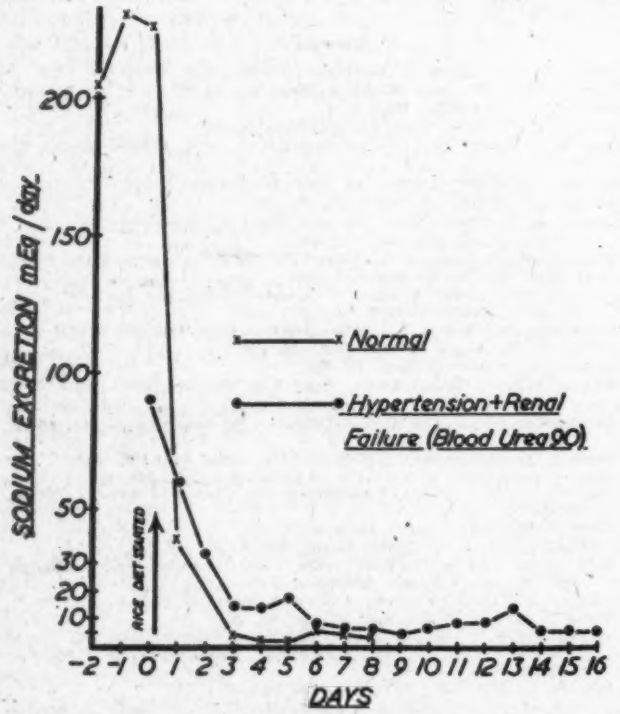

FIG. D.—Effect of rice diet on sodium excretion in a normal person and in one with renal failure (see text).

Now in renal failure the most remarkable fact is that, whereas the plasma level of urea, creatinine, and phosphate (and many other substances such as sulphate, uric acid, and indican) is much increased, the plasma content of sodium and potassium remains strictly within normal limits. This is obviously of great importance to the survival of the patient, for the body will tolerate only very small variations in the concentration of sodium and potassium in its extracellular fluid. It is true that cases of sodium deficiency resembling Addison's disease have occasionally been encountered in renal failure (Thorn, Koepf, and Clinton, 1944), and that in states of anuria and oliguria fatal retention of potassium may occur (Keith and Burchell, 1949), but so long as the kidney continues to excrete water, sodium and potassium plasma levels generally remain normal until the last few days or hours of the patient's illness.

Since the glomerular filtration rate in renal failure is much reduced, retention of sodium and potassium would certainly occur if tubular reabsorption continued as before. Considered teleologically, therefore, the only thing that the kidney can do to prevent this retention is to reduce tubular reabsorption of sodium and potassium, and this is precisely what happens.† I have shown elsewhere (Platt, 1950) that there is a close relationship between the degree of renal failure as indicated by the plasma creatinine level and the percentage of filtered sodium and potassium which is rejected by the tubules and excreted in the urine. I will not repeat all the evidence here, but will give an illustration of what happens in normal and in renal failure subjects.

	Normal		Renal Failure
Plasma sodium	140		140 mEq/l.
Glomerular filtration rate	100		(say) 20 ml./min.
Sodium load filtered	14		2·8 mEq/min.
Amount reabsorbed (99%) =	13·86		= (95%) 2·66 mEq/min.
Remainder excreted (1%) =	0·14		= (5%) 0·14 mEq/min.
which is equivalent to	11·8		11·8 g. NaCl/24 hours

You will see from this table that the plasma sodium remains normal in spite of a glomerular filtration rate which is reduced from the normal of about 100 ml. per minute to 20. By reabsorbing only 95% instead of 99% of the sodium reaching them, the tubules reject for excretion as much sodium as before. That this is a physiological adaptation to prevent sodium retention and not the fortuitous effect of damaged tubules no longer able to reabsorb as much sodium as before is, I believe, demonstrated by three facts: (1) That the plasma sodium remains strictly normal for months or years in chronic cases of renal failure. (2) The rats from which we have removed 80% of their renal tissue (Platt, Roscoe, and Smith, 1952) show the same adaptation as the patients, reducing their tubular reabsorption of sodium to meet

*The second of two Lumleian Lectures delivered to the Royal College of Physicians of London on April 1 and 3, 1952.

†The only alternative method of keeping the plasma sodium normal would be to retain both sodium and water in equivalent proportions. This happens in oedematous nephritis but not in renal failure.

the circumstances and prevent sodium retention. (3) The failing kidney can, when required, still conserve sodium.

Fig. D also shows a hypertensive uraemic patient on the rice diet. His blood urea was 90 mg. per 100 ml. It will be seen that he was still able to reduce his sodium excretion to negligible amounts, though it took him longer than the normal person to do so. His tubules were finally reabsorbing about 99.8% of the sodium reaching them.

In the case of potassium I showed (Platt, 1950) that in renal failure not only do the tubules reject a greater fraction of the load delivered to them by filtration, but in severe cases the rate of excretion may actually exceed the rate of filtration of potassium; in other words, the potassium clearance may exceed the creatinine or inulin clearance. This can only mean that a mechanism exists for the active tubular excretion of potassium. Leaf and Camara (1949) found the same phenomenon in chronic renal failure, and McCance and Widdowson (1937), in a case of renal failure due to alkalosis, reported a potassium clearance temporarily greater than that of inulin. Berliner and Kennedy (1948) have shown that tubular excretion of potassium may be induced in normal dogs under special circumstances. We have since found the same phenomenon in experimental renal failure in the rat. One of our rats, whose blood urea rose to 636 mg. per 100 ml., had a potassium clearance more than twice that of his creatinine clearance.

This demonstration of the active tubular secretion of potassium is another indication that the remaining remnant of renal tissue, both in experimental and in human renal failure, can perform extraordinary feats. The renal tubules behave not as a machine failing and giving out but as a mechanism pushed to the limit of its capacity. It is only in the study of human disease that such adaptations are revealed; and having been revealed in the extremes of disease, they may disclose a normal mechanism hitherto unsuspected.

We may briefly summarize this section by saying that excretion of sodium and potassium is efficiently maintained in renal failure, first by the simple device of the remaining tubules reabsorbing a smaller proportion of these substances, and, secondly, in the case of potassium, by a process of active tubular excretion.

2. Phosphate and Calcium

The anion phosphate does not behave like the cations sodium and potassium in renal disease, for it is retained by the kidney and may reach high levels in the blood. As with urea, the high plasma level facilitates the excretion of phosphate, and the phosphate clearance approaches but does not exceed that of inulin or creatinine (Platt, 1950).

In calcium metabolism we have an excellent example of a train of adaptations which can be set in motion by renal failure. The usual tendency is for the plasma calcium to fall, and several factors are concerned in this. One is the high plasma phosphate level which, in accordance with the usual reciprocal relationship between calcium and phosphate, will tend to depress the calcium level. A high plasma phosphate probably also interferes with calcium and phosphate absorption from the intestines. A third factor probably contributing to calcium depletion is acidosis, if calcium is used as a base with which to excrete acid radicals.

There is need for further work in assessing the relative importance of the several factors involved.

Whatever be the cause or causes of the low plasma calcium, it appears to act as a stimulus to the parathyroids (Stoerk and Carnes, 1945; Albright and Reifenstein, 1948), and these glands are found to be hyperplastic in renal failure (Pappenheimer and Wilens, 1935). The excess of parathyroid hormone tends to restore the plasma calcium towards normal by mobilizing calcium from bone (Follis, 1950; Milne, 1951). This adaptation, while preventing tetany and other ill effects of calcium deficiency, has a side-effect in cases of renal failure of long duration, especially in young persons, in causing the bony changes of hyperparathyroidism to appear, which Albright and Reifenstein have called "renal osteitis fibrosa" (Fig. E). It should be

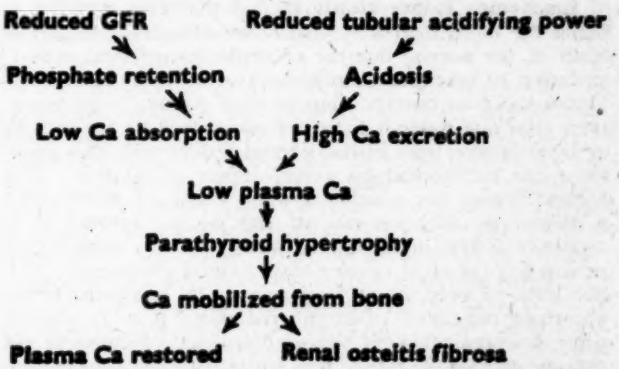

FIG. E.—Scheme showing the effect of renal failure on calcium metabolism (modified from Albright and Reifenstein, 1948).

mentioned that the older term "renal rickets" is not entirely a misnomer, since Follis (1950), by careful histological examination, has shown that changes typical of rickets or osteomalacia may also be found. Thus there would seem to be a factor interfering with the deposition of calcium and phosphorus in bone in addition to the parathyroid action favouring bone resorption. In all probability the acidosis itself both contributes to the mobilization of calcium from bone and interferes with its deposition. The bony changes can sometimes be reversed by a combination of low phosphorus diet, alkali therapy, and calciferol.

It is extraordinary how promptly parathyroid hypertrophy occurs, at any rate in the rat. Our rats' parathyroids showed clear evidence of hyperplasia with mitoses within 14 days of removal of their renal tissue (Platt, Roscoe, and Smith, 1952).

It would be impossible in the time at my disposal, and it would in any case be tedious, to attempt a complete survey of what is known of functional adaptation in renal disease. For instance, the kidney's defence against acid accumulation in the body is put to a severe test, and yet clinical and biochemical evidence of acidosis is generally lacking until the final stage of disease. It is known that the failing kidney cannot produce a highly acid urine, nor can it produce ammonium ion in adequate amount to neutralize the anions it must excrete; but these aspects of our subject and the excretion of bicarbonate in renal failure are as yet inadequately investigated, and I have no new observations to offer.

The Excretion of Water

The rate of water excretion seems to be determined in normal individuals by two main factors. The first is the solute load: we have already seen that there is a certain minimum rate of urine flow which is necessary for the excretion of dissolved substances, and when solutes are present in excess, as in the clinical state of diabetes mellitus, the amount of water excreted increases very greatly. This type of polyuria is known as osmotic diuresis because the increased osmotic pressure of the tubular fluid prevents the reabsorption of water which is believed to take place in the proximal tubule. Even under normal circumstances the amount of water we excrete is largely determined by the

solute load and especially by the varying need to excrete electrolytes (Stanbury and Thomson, 1951), since the rate of excretion of urea is much more constant.

In renal failure, as we have already seen, the rate of excretion of all urinary constituents such as urea, sodium, potassium, phosphate, and chloride remains remarkably normal, and so the rate of water excretion must remain at least normal also, though actually it increases because of the inability of the failing kidney to produce a concentrated urine. This inability to concentrate we have discussed in the first lecture, where it was pointed out that the excretion of a normal quantity of urinary solutes by a fragment of functioning kidney clearly implies that each nephron is doing far more than a normal share of work. If only a tenth of the normal number of nephrons remains, each is excreting 10 times its normal quota of solutes and so must behave as if an osmotic diuresis were constantly in operation. Far less water is therefore reabsorbed by the tubules in renal failure than in the normal kidney, and this statement can be checked by a very simple calculation. If a normal person has a urine flow of 1 ml. per minute and a glomerular filtration rate of 100 ml. per minute he is reabsorbing 99% of his filtered water; but if a renal failure patient has the same urinary output and a glomerular filtration rate of only 10 ml. per minute he is obviously reabsorbing only 90% of his filtered water, and the rate of urine flow per tubule is increased tenfold. Because of the osmotic diuresis the rate of flow will actually be even greater. This is probably related to the tubular dilatation, which is, as we have seen, such an obvious structural feature of the failing kidney. The polyuria of renal failure is of course accompanied by an increased water intake and by conscious thirst in severe cases.

The second determinant of water excretion comes into operation when water itself is present in excess. This is mediated, as Verney (1947) has shown, by the osmoreceptors, which inhibit the secretion of pituitary antidiuretic hormone.* There are thus two types of diuresis: osmotic diuresis in which the urine approaches the concentration or osmolarity of the plasma; and water diuresis, best seen after the ingestion of a litre or more of water, in which the urine becomes extremely hypotonic, approaching the osmolarity of distilled water (Frey and Frey, 1950).

According to Wolf, the Sioux Indians used water diuresis as an alarm clock, adjusting the dose of water to be drunk before retiring according to the time they wished to go out on the war trail in the morning. I suppose that they worked it out empirically, unless they were aware that the amount of the diuresis is approximately proportional to the logarithm of the time (Wolf, 1950).

It has long been known that the failing kidney cannot produce an efficient water diuresis, and this, of course, is the basis of the Volhard dilution test.

Water Diuresis

Fig. F shows the effect that drinking 1,100 ml. of water had on myself and on a patient with severe renal failure whom Dr. Roscoe and I investigated and whose blood urea at the time of the test was 290 mg. per 100 ml.

In the normal subject the water output rose sharply to 16 ml. per minute, a highly efficient but socially inconvenient response, whereas the patient reached a urine flow of only about 4 ml. per minute. Thus the speed of getting rid of a water load of this magnitude is much reduced in renal failure. The same thing occurs in rats deprived of renal tissue (Platt, Roscoe, and Smith, 1952). The graph also shows what again is well known, that water diuresis is not brought about by increasing the rate of filtration at the glomerulus, but (on current theories) by reducing the amount

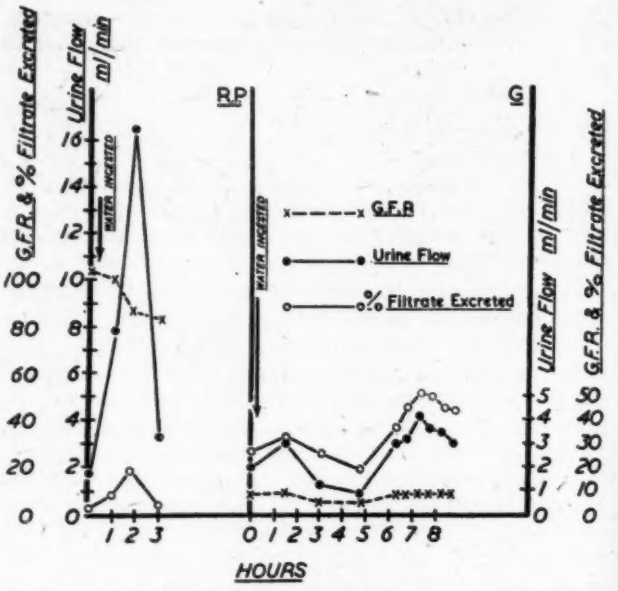

FIG. F.—Water diuresis in a normal subject and in renal failure (see text).

of water reabsorbed in the tubules. It shows further that in spite of the ingestion of this large amount of water about 80% of the filtered water is still being reabsorbed in the normal kidney, thus confirming Homer Smith's (1937) assertion that the reabsorption of 80% or more of the filtered water is obligatory. But in the case of the patient, the filtration rate (measured by both inulin and creatinine clearance) was only about 8 ml. per minute, and at the height of his diuresis he reached a urine volume of 4.16 ml. per minute, or about 50% of his filtered water, thus upsetting normal conceptions of water reabsorption rather badly: another example of the value of studying physiological mechanisms when they are contending with the stress of disease. Some of this effect was due to osmotic diuresis, but much was due to water ingestion, because the patient's urine at the height of the diuresis was only about half isotonic with regard to sodium, containing 70 mEq of sodium per litre.

Now, in order to produce a hypotonic urine in respect of sodium (or some other solute), obviously a *hypertonic* solution must be reabsorbed by the tubules. Given the filtration rate and the plasma and urinary concentrations of sodium, the sodium content of the reabsorbed fluid can be calculated, the calculation being:

$$\frac{\%\ \text{reabsorption of sodium}}{\%\ \text{reabsorption of water}} \times \text{plasma sodium}$$

Because of the very low glomerular filtration rate, it is much more difficult for the patient to secrete a hypotonic urine than for the normal subject, and a much more hypertonic fluid has to be reabsorbed by the tubules. Calculation shows that, in order to produce the hypotonic urine which was actually found in this case, a strongly hypertonic solution (216 mEq Na per litre) must have been reabsorbed all the way along the renal tubule, and that the renal tubular function would have had to be much more efficient in this respect than has ever been demonstrated in a normal subject.

While this is not impossible, we must remember that the whole basis of the calculation is the assumption that water diuresis is purely a question of filtration and reabsorption; and that assumption, which is currently accepted by renal physiologists, I would like seriously to question. If we postulate instead a mechanism for the tubular secretion of water, or more probably of a hypotonic fluid, the observed facts can be explained much more simply, and, I think, in a manner more consistent with what we know of physio-

*It should be noted in passing that the adrenal cortex is in some way necessary to water diuresis, since patients with adrenal insufficiency cannot excrete water efficiently; but this faculty is restored by the administration of cortisone (Oleesky and Stanbury, 1951).

logical processes. Such a theory would have three advantages as an explanation for the observed facts. First, it would avoid the necessity for assuming that the renal failure patient is capable of reabsorbing a much more highly concentrated solution than the normal, and, although it assumes as an alternative that some part of the renal tubule can secrete a very hypotonic fluid, it is assumed that this is a function also present in the normal kidney. Secondly, it would avoid the assumption that a renal failure patient reabsorbs only 50% of his filtered water whereas in extreme conditions of diabetes insipidus plus osmotic diuresis the normal kidney still reabsorbs about 80% (Brodsky and Rapoport, 1951). Thirdly, assuming that the increased urine volume during water diuresis was due to tubular secretion of water, in our experiment we could conclude that the normal subject can secrete about 15 ml. of water per minute, whereas the renal failure patient secretes only about 2 ml. per minute—a fraction consistent with the amount of functioning renal tissue which he probably possessed.

You might well ask why, if this is the case, the kidney should perform its functions in such diverse ways; but if tubular secretion of potassium occurs, why not water secretion? And let me remind you that although the kidney is a highly efficient organ the evidence of comparative anatomy, of embryology, and of evolution suggests that it has developed in what would seem to be a somewhat haphazard manner. The kidneys of certain marine teleosts, although they have no glomeruli at all, can yet secrete a hypotonic urine (Marshall, 1930), and it seems to me not beyond reason that this function has been passed on to the mammalian kidney.

Since we elaborated this theory, Brodsky and Rapoport (1951) have also concluded from their studies on osmotic diuresis in subjects with diabetes insipidus that tubular secretion of water is more consistent with the facts and less extravagant in renal osmotic work than water excretion solely by filtration and reabsorption. Dr. Roscoe is at present making further studies of this question, but allows me to say that she has now made the very striking observation that in two cases of terminal renal failure she has found a serum creatinine concentration considerably higher than that of the urine. Since tubular reabsorption of water can only concentrate the urine and not dilute it, this would be proof of water secretion, if we were quite sure that creatinine clearance was a true measure of the glomerular filtration rate. Unfortunately we cannot be certain of this, and there are known fallacies in the estimation of serum creatinine (Lauson, 1951). Despite these uncertainties this would seem to be very strong evidence in favour of water secretion. If it does occur we do not, of course, know whether it is a function of the proximal or the distal tubule, or perhaps of that evasive structure, the loop of Henle.

A few practical inferences can be drawn from our consideration of the excretion of solutes and water in renal failure.

The kidney is performing a minimum amount of work when it produces a urine the composition of which differs as little as possible from that of the plasma. The salt content of urine is usually less than that of plasma, whereas the urea content is normally much greater. Therefore we must restrict protein intake to reduce urea excretion, but must give an adequate, in fact a liberal, ration of salt. The failing kidney needs a generous supply of water for its excretion of solutes, but has difficulty in getting rid of water in excess. In determining water intake the instinctive demands of the patient, regulated as they are by an extremely efficient thirst mechanism, are usually the most accurate guide.

Hypertension in Renal Failure

Before discussing my final subject, which is renal anaemia, I will include a few words on the relation of hypertension to renal disease. I cannot consider it here in detail, and there is so much unsettled controversy that it is a subject which must either be dismissed very briefly or be discussed at great length.

The fact that chronic renal disease is very often (though not constantly) associated with hypertension, and the fact that the glomerulus is primarily dependent upon the blood pressure for its function of filtration, suggest that hypertension is an adaptation called into play to raise the glomerular pressure. Goldblatt's (1934) classical experiments seemed to confirm this by showing that reduction in renal blood flow had hypertension as its inevitable sequel. Further work, however, has led to many complications and little clarification. The concept of a simple relationship between blood pressure and renal blood flow is difficult to maintain in view of the experiments of Grollman, Turner, and McLean (1951), who showed that dogs kept alive by peritoneal irrigation after total nephrectomy developed severe hypertension.

The subject has not been much illuminated by the continual confusion in the work of some authors (e.g., Goldring and Chasis, 1944; Homer Smith, 1951) between essential hypertension, the origin of which is still unknown, and renal hypertension, which would seem to be clearly related to pre-existing unilateral or bilateral disease of the kidneys. One thing seems certain from the work of Wilson and Byrom (1941) and Byrom and Dodson (1948)—namely, that whatever the origin of the hypertension, even if it be at first adaptive and beneficial to survival, it can reach a stage when a vicious circle sets in and further elevation of blood pressure leads to arteriolar necrosis with serious and rapid deterioration in renal function. This is a syndrome often encountered when severe hypertension and renal disease coexist.

The Anaemia of Renal Failure

My last section is devoted to the consideration of a condition which is invariably present in renal failure—namely, anaemia. Its origin has long been a mystery, and, though I shall bring forward a new hypothesis concerning its causation, I cannot claim that the mystery is yet solved.

First of all I would like to set out some of the known facts, which are as follows:

1. All cases of renal failure with prolonged nitrogen retention, whatever the cause, are accompanied by anaemia. The anaemia is independent of the presence or absence of hypertension.

2. The anaemia is normochromic in type. In my department, 84 observations on 49 patients showed a colour index between 0.85 and 1.07 in all but one instance, although the range of haemoglobin varied from 26% to 96% (3.85 to 14.2 g. per 100 ml.). The exception showed a colour index of 0.81.

3. The haemoglobin level is quantitatively related to the blood urea, falling as the blood urea rises in such a manner that each 50 mg. rise of urea per 100 ml. blood is accompanied by a fall of about 2 g. per 100 ml. in haemoglobin (Roscoe, 1952).

4. The quantitative relationship is disturbed in cases where the blood urea is rising very rapidly, changes in haemoglobin lagging behind those of blood urea, but with this exception—the haemoglobin does not depend upon the duration of the uraemia, for patients whose blood urea remains at a fairly constant high level for months (or in rare cases for years) do not show a progressive fall in haemoglobin.

5. The same type of anaemia may occur in extrarenal uraemia of long duration (e.g., pyloric stenosis), and I have seen it occur in uraemia from a spinal injury with urinary retention and overflow incontinence. Severe anaemia also occurs in nephrectomized dogs kept alive by peritoneal lavage (Grollman, Turner, and McLean, 1951).

6. The anaemia is not due to haemodilution, since the blood volume is usually normal or low in these cases (Harris and Gibson, 1939).

7. It is not due to haemorrhages, as even haematuria is nearly always absent in chronic cases of renal failure. A haemorrhagic stage of uraemia may occur terminally, with epistaxis and bleeding gums, but the anaemia is well established long before this stage is reached.

8. There is usually no clear evidence of increased blood destruction. Most cases show no increase of serum bilirubin and no increased excretion of urobilinogen (Loge, Lange, and Moore, 1950). Investigation of the survival of transfused cells by the Ashby technique showed a normal survival in one case observed by Dr. M. D. Milne in my department, but Emerson and Burrows

(1949) claim to have shown an increased rate of destruction of transfused cells. Loge, Lange, and Moore (1950) summarize their investigations by saying that red-cell survival is diminished only in severe cases with gross azotaemia. In these terminal cases reticulocytosis may be found, as we have also observed.

9. Most observers state that the mechanism of the anaemia is depressed bone-marrow function, but according to Callen and Limarzi (1950) the marrow, which is always normoblastic, may be hypercellular—the defect, they believe, being one of delivery rather than of maturation.

10. The anaemia responds neither to iron nor to liver extract. Utilization of radioactive iron administered intravenously is decreased. Serum iron and erythrocyte protoporphyrin show no characteristic pattern, unlike the anaemia of chronic sepsis (Loge, Lange, and Moore, 1950).

The main conclusion we may draw from these facts is that in chronic renal failure an anaemia regularly develops which varies in severity with the degree of azotaemia, and is due to depression of bone-marrow function or non-delivery of red cells from marrow to peripheral blood. Stanbury (personal communication), however, points out that while the survival rate of transfused cells may be normal, the rate of destruction of the patient's own cells has not been investigated. A slowly developing anaemia due to increased blood destruction might not show a measurable degree of hyperbilirubinaemia, especially if hepatic function is normal.

The fact that there exists a correlation between the severity of the anaemia and the degree of urea retention does not of course mean that the high blood urea is directly responsible for affecting the marrow, but it does suggest, perhaps, that some retained metabolic product is concerned. If so, three obvious possibilities are urea, creatinine, and phosphate. A high plasma phosphate might interfere with iron absorption from the intestine or with iron transport, but in such a case one would expect the anaemia to be hypochromic. Moreover, a high serum phosphate occurs in hypoparathyroidism, and is not associated with anaemia (Milne, personal communication; Drake, Albright, Bauer, and Castleman, 1939; Albright and Ellsworth, 1929; Albright, Burnett, Smith, and Parson, 1942).

To see if urea would depress the haemoglobin level I took from 30 to 45 g. of urea daily for 16 days, during which time my blood urea was between 50 and 54 mg. per 100 ml. on waking, and 70 and 90 mg. during most of the day. This had no effect whatever upon the haemoglobin, and the experiment was given up because it was only with difficulty compatible with a day's work, the high blood urea producing a dry mouth, thirst, lethargy, and a tendency to headache. Incidentally it had no noticeable effect on weight and appetite, on blood pressure, or on serum sodium concentration or creatinine clearance. By calculation 16 days with a blood urea in this range would be the minimum that might possibly be expected to show some alteration in serial haemoglobin levels estimated by the alkaline haematin method, and although the experiment was negative it was not pursued long enough to disprove finally a connexion betwen urea and haemoglobin.

I next tried creatinine in doses of 3 to 4 g. per day for 29 days, sufficient to produce a serum creatinine of 1.8 mg. per 100 ml. in the morning and nearly 5 mg. later in the day. This also had no effect whatever on the haemoglobin level.

So far, then, we have failed to find the substance responsible for depression of haemoglobin in renal anaemia.

There is, however, another approach to the problem. Most authors consider that the anaemia of renal failure is evidence of a state of uraemic poisoning; that it is something undesirable. Yet restoration of haemoglobin towards normal or even up to normal values has never been shown to have any beneficial effect upon renal function in these cases (Emerson and Burrows, 1949), and it may even prove harmful (Salvesen, 1948).

It has occurred to me that the anaemia may itself be a process of adaptation, and this is my justification for discussing it in these lectures. If the renal blood flow is limited in quantity, as it appears to be in renal failure (Earle, Taggart, and Shannon, 1944; Corcoran, Taylor, and Page, 1948) it is obviously to the advantage of glomerular filtration that the blood should contain more than its normal quota of plasma. That the renal plasma flow is carefully regulated in the normal kidney is suggested by studies of anaemia (Bradley and Bradley, 1947; Paterson, 1951) and of polycythaemia (de Wardener, McSwiney, and Miles, 1951). These show that when the haematocrit level is reduced, as in anaemia, the total renal blood flow is diminished but the plasma flow remains approximately normal. An increased haematocrit level, as in polycythaemia, results in an increase of total blood flow with some reduction in plasma flow. Paterson also showed that in experimental anaemia the plasma filtered at the glomerulus bore a direct relationship to the amount of plasma presented to it. Representing renal blood flow by RBF, plasma flow by RPF, and haematocrit level by H, we have an approximate relationship: $RPF \propto \frac{1}{H}$ if RBF is constant; $RPF \propto RBF$ if H is constant. Whence $RPF \propto \frac{RBF}{H}$, so that if RBF is reduced as in renal failure, a corresponding reduction in haematocrit level is necessary to keep the plasma flow as high as possible. If this is associated, as in other cases of anaemia, with an increased cardiac output, renal plasma flow will be even more favourably affected by the development of anaemia.

The fact that in chronic non-progressive cases of renal failure the anaemia reaches a certain degree dependent upon the severity of uraemia, and then remains constant, strongly suggests that a mechanism may exist for the establishment of anaemia in order to conserve the renal plasma flow.

As I remarked at the beginning of these lectures, teleological arguments of this type cannot take us beyond the stage of hypothesis, but further evidence might be obtainable by investigating various aspects of renal function, before and after the transfusion of renal failure patients, in more detail than in the studies already made by Salvesen (1948). Obviously such an investigation is both desirable and justifiable, since transfusion is a therapeutic measure often advised in these cases, and although it improves the general well-being of the patient it is important to know whether it acts favourably or otherwise upon his kidneys.

I had thought to have these studies ready for inclusion in these lectures, but suitable cases are not easy to find. Many of our renal failure patients are in the terminal phase of their illness, when renal function often deteriorates rapidly for no obvious reason and when estimations such as the clearance of *para*-amino-hippurate probably give quite unreliable information on renal plasma flow. Many of the patients, moreover, have severe hypertensive symptoms, which would in my view make transfusion unwise. Stationary or slowly progressive cases of renal failure without hypertension do not present themselves at the convenience of Lumleian lecturers, so the subject must be left for study in a more leisurely manner.

Even if the hypothesis that the anaemia of renal failure is an adaptation ensuring maximum renal plasma flow is acceptable, it must still be mediated by some factor affecting the bone marrow or the red cells themselves. This factor does not appear to be urea, creatinine, or phosphate. It may be some other retained product. Further search for the anaemia-producing factor would seem to be a profitable investigation to pursue, for if it could be found we might by good fortune discover a factor concerned with the physiological regulation of the volume of red cells in the peripheral blood.

Conclusion

I have tried to show that most if not all the functional disturbances known to occur in human renal failure are precisely those which occur in animal experiment as a result of reduction in the amount of functioning renal

substance—that is, loss of nephrons. In such experiments the remaining nephrons enlarge and take on a volume of work which in the normal kidney they are never called upon to perform. The same, I believe, occurs in human disease, and our concept of renal failure should not be one of disordered function, but rather one of extremely efficient function by a renal remnant now too small for its task. I have tried to describe some of the ruses and tricks which the remaining renal substance employs to carry on its work so as to maintain excretory function and to preserve as long as possible the composition of the *milieu intérieur* with regard to sodium, potassium, water, and other substances essential to survival.

I have pleasure in acknowledging the great debt I owe to members of my team in Manchester and formerly in Sheffield for their help, discussion, and criticism. I have referred to them previously as my fellow nephrophiles, and the term is not entirely facetious, for I would like my envoy to be a salutation to the kidney itself—*viscus elegantissimum*, as Jean Oliver, quoting from the older anatomists, has recently called it. For the kidney has contributed much interest to my life and thought for many years; I would like to express my admiration of its structure and for the quiet and efficient way in which it handles its problems; and although we are still too ignorant to do much to remedy its disorders, these, I feel, are not the fault of the kidney itself, which seems to devise every possible adaptation to overcome the handicaps imposed upon it by disease.

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SYMPATHECTOMY FOR RAYNAUD'S DISEASE

RESULTS OF GANGLIONECTOMY AND PRE-GANGLIONIC SECTION COMPARED

BY

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Sympathetic ganglionectomy for Raynaud's disease of the hands produced good results in most cases, but relapse sometimes followed it (Ross, 1935). Telford (1935) and Smithwick (1936) suggested that relapse might be avoided by carrying out a purely pre-ganglionic section of the sympathetic chain. The two operations have never been adequately compared. The present study was planned so that a direct comparison could be made between a group of patients who had had pre-ganglionic section and another earlier group treated by ganglionectomy and reported upon by Ross in 1935. The severity of the disease and the results of operation were assessed by the same methods in each series. The results are compared in Table I.

The patients who had undergone pre-ganglionic section were followed for one to thirteen years after operation. All had been operated upon by members of the surgical professorial unit at St. Bartholomew's Hospital. They had been examined at intervals since operation and again for the purpose of this review,

TABLE I.—*Results of Different Operations for Raynaud's Disease Compared*

Operation	No. of Patients	Results		
		Success*	Improved†	Failure‡
Ganglionectomy (1935 series)	41	29 (71%)	4 (10%)	8 (19%)
Pre-ganglionic section (series to 1951)	39	27 (69%)	5 (13%)	7 (18%)

* Attacks less frequent and less severe. No recurrence of ulceration.
† Ulcers recurred, but pain less and working capacity increased.
‡ No change or worse.

except a few who lived too far away and who replied by letter to a questionary. The results of the two operations are very much alike. The two groups are comparable in size and in other respects, except that many of the patients in the second series have been followed for a longer time. It might be that this allowed more failures to become manifest and showed pre-ganglionic section in a less favourable light. To test that possibility the records of the patients who had been followed for more than two years after pre-ganglionic section and finally assessed as failures were examined to see whether there was any evidence to suggest that the results might have been different if they had been assessed at an earlier date. There were five such patients, and in four of them it was quite evident from the case records that the result was a failure before a year had passed after operation. Information was not available about the early progress of the other patient.

Types of Pre-ganglionic Operation Compared

The pre-ganglionic operations were done in one of two ways: (*a*) Telford's method, in which the sympathetic chain was divided below the third ganglion and turned upwards after the rami communicantes of the second and third ganglion had been cut. (*b*) A modification of Smithwick's method in which pre-ganglionic section was performed as in (*a*) but in addition the roots of the second and third thoracic spinal nerves were cut just outside or inside the dura mater.

The results of the two types of operation are shown in Table II. There is little difference between them. This

TABLE II.—*Pre-ganglionic Operations Compared*

Operation	No. of Limbs	Results		
		Success	Improved	Failure
Telford	43	30 (70%)	7	6
Smithwick	31	22 (68%)	2	7

suggests either that the additional measures taken in the Smithwick technique to prevent regeneration were ineffective or else that relapse was due to some other cause than regeneration.

Factors Influencing the Prognosis after Operation

In Table III are compared the results of ganglionectomy and pre-ganglionic section in three grades of severity of the disease—uncomplicated cases and those complicated by ulceration or scleroderma.

TABLE III.—*Analysis According to Severity of Disease*

Operation	Severity	No. of Patients	Results		
			Success	Improved	Failure
Ganglionectomy (1935 series)	Uncomplicated	21	21	—	—
	With ulceration	9	7	2	—
	With scleroderma	11	1	2	8
Pre-ganglionic section (1951)	Uncomplicated	22	17	1	4
	With ulceration	10	5	4	1
	With scleroderma	7	5	—	2

The patients with scleroderma treated by pre-ganglionic section (two failures out of seven) did better than those treated by ganglionectomy (eight failures out of eleven), but much significance should not be attached to this, because the numbers are small. Otherwise there is no evidence that the initial severity of the disease influenced the result after operation.

Other factors which might have affected the result after operation were: the age of the patient when the disease started; the duration of the disease before operation; the greater severity judged by involvement of the feet as well as the hands; and the length of the initial period of complete freedom from vasoconstrictive attacks after operation. These factors were studied in the patients who had pre-ganglionic section, but were found to have no effect upon the ultimate results.

Discussion

The most striking finding is the similarity between the results of ganglionectomy and pre-ganglionic section in two comparable groups of patients. It is interesting to see what light this casts on the recurrence of vasomotor attacks after operation. Several theories have been put forward to account for this recurrence after sympathectomy for Raynaud's disease. They include: (1) development of sensitivity of the denervated vessels to circulating adrenaline; (2) regeneration of the divided sympathetic nerves so that continuity is restored; (3) reorganization of alternative nerve pathways; and (4) persistence of a local fault in the digital vessels.

Experimental work has shown that sensitivity to circulating adrenaline is greater after ganglionectomy than after pre-ganglionic section. If adrenaline sensitivity were the explanation of the recurrences after operation the results of pre-ganglionic section would be expected to be better than those of ganglionectomy. It has also been objected to as an explanation on other grounds. Simmons and Sheehan (1937) pointed out that the effects of sensitivity would be expected to appear early, whereas the relapses do not appear for some weeks or months after operation.

Regeneration has been frequently suggested (Simmons and Sheehan, 1937; Smithwick, 1940; White and Smithwick, 1944; Haxton, 1947) as an explanation of relapse. If ganglionectomy has been done it is necessary to postulate the growth of pre-ganglionic cholinergic medullated axons down the degenerated remains of the adrenergic non-medullated post-ganglionic fibres. There is no evidence that this is possible. Kirgis *et al.* (1950) have shown that regeneration does not take place in the cervical sympathetic chain of the cat after ganglionectomy, although it may do so after pre-ganglionic section.

Additional measures taken to prevent regeneration in the pre-ganglionic operations made no difference to the results, as the comparison of the Telford and Smithwick operations showed. If regeneration were the cause of relapses they should not occur at all after ganglionectomy, and they should be seen less often after Smithwick than after Telford operations. As these operations give similar results some other explanation must be sought.

Reorganization of alternative pathways has been suggested (Ray *et al.*, 1943). It is known from anatomical studies that sympathetic fibres and so-called intermediate ganglia exist in the cervical region, where they are outside the scope of surgical removal (Skoog, 1947; Boyd and Monro, 1949). Pathways such as these might in time become the mediators of recurrent nervous vasomotor activity. A more acceptable explanation is that put forward by Lewis (1946). He considered that the disease and its recurrence after sympathectomy were due to continued existence of a "local fault" in the digital vessels.

What regulates the intrinsic tone of the vascular smooth muscle in the absence of vasomotor nerves is unknown. Barcroft and Walker (1949) have shown that it recovers rapidly after sympathectomy even in normal vessels. Until more is known of the fundamental physiology of vascular smooth muscle the nature of the "local fault" in Raynaud's disease cannot be determined.

Finally, it must be decided which operation is best for the patient. Cervico-thoracic ganglionectomy has certain disadvantages, such as the production of Horner's syndrome and nasal congestion. Pre-ganglionic section avoids these and produces equally good effects upon the digital vessels. Smithwick's posterior approach through the third rib is easier and safer for the less experienced operator but sometimes gives persistent post-operative pain in the wound. For the surgeon familiar with the anterior route, Telford's operation is more suitable.

Conclusions

The clinical results of ganglionectomy and of preganglionic sympathetic section for Raynaud's disease are the same.

The absence of unpleasant side-effects from preganglionic section makes it preferable to ganglionectomy.

The similarity in results of the two operations suggests that nerve regeneration is not the chief cause of relapse after sympathectomy.

The possible causes of relapse are discussed. The "local fault" postulated by Lewis is thought to be important.

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SEPTIC JAUNDICE

A COMPLICATION OF PERITONITIS FOLLOWING GASTRO-INTESTINAL SURGERY

BY

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The occurrence of jaundice shortly after operation gives rise to anxiety, and the determination of its cause becomes a matter of importance. That jaundice can be caused by sepsis is known, but reports of this type of case are rare. Brown and Counsellor (1932) described two cases of acute appendicitis in which jaundice was present before operation. An appendix abscess was present in one, and in the other the organ was gangrenous and purulent. With the subsidence of infection after operation the jaundice cleared rapidly. Philipowicz (1934) reported the development of deep jaundice due to an enormously distended and inflamed caecum and appendix in a 73-year-old man, with no obvious liver or gall-bladder disease at operation. The jaundice receded rapidly after operation. Simeone and Stewart (1940) described two cases of peritonitis after appendicectomy in which jaundice developed. In both cases haemorrhagic manifestations and evidence of liver failure occurred and both terminated fatally. Post-mortem examination, allowed in only one case, showed a diffuse central lobular necrosis of the liver. Only in one of many textbooks consulted is jaundice mentioned as a complication of peritonitis. Muir (1941) states that areas of necrosis may occur in the liver in acute peritonitis, but makes no mention of jaundice.

The six cases described below appear to fall into one category, for which the descriptive title of septic jaundice is suggested. The term "infectious" or "infective jaundice" carries the risk of confusion with infective hepatitis of viral origin, and "toxic jaundice" has been applied to jaundice caused by so many diverse agents that it lacks specificity.

Clinical Description

Details of the cases are shown in Tables I to IV. The histological findings in two further fatal cases of peritonitis following gastrectomy are included in Table IV for purposes of comparison. These two cases did not develop jaundice.

It will be seen that in all cases the operation involved the gastro-intestinal tract, and that post-operative complications were severe. Peritonitis, local or general, occurred in every case. Wound breakdown, subphrenic and intraperitoneal abscesses, metastatic abscesses, faecal fistulae, abdominal wall abscess, and vesico-perineal fistula occurred, necessitating secondary operation in most cases. Unfortun-

TABLE I.—*Pathological and Nutritional States*

Case	Sex	Age	Pathological State	Nutrition	Diet
1	F	43	Carcinoma of stomach	Fair	Gastric
2	M	44	Gastric ulcer	,,	,,
3	M	51	Diverticulitis (localized)	Good	Normal
4	M	36	Gastric ulcer	Fair	Gastric
5	M	58	Carcinoma of rectum	Good	Normal
6	F	35	Intestinal obstruction due to multiple adhesions	,,	,,

TABLE II.—*Details of Operations, Complications, and Treatment.*

Case	Operation	Septic Complications	Jaundice: Onset and Duration	Transfusions*	Sulphonamides†
1	13/2/50: abdomino-thoracic total gastrectomy	16/2/50: ? peritonitis 22/2/50: drainage of peritonitis and right subphrenic abscess	17/2/50: jaundice till death on 24/2/50	13/2/50 (3) 17/2/50 (3)	23/2/51: sulphadimidine 2 g. I.M.
2	29/10/49: subtotal gastrectomy	2/11/49: Stomal obstruction—moderate 6/11/49: laparotomy—localized peritonitis and leak from suture line 10/11/49: multiple septic pulmonary infarcts 11/11/49: hypopyon	7/11/49: jaundice till death on 14/11/49	6/11/49 (1)	11–14/11/49: sulphatriad 17 g.
3	17/11/50: partial colectomy, end-to-end anastomosis	26/11/50: faecal fistula in wound 29/11/50: resuture of wound 5/12/50: jejunostomy for intestinal obstruction	4/12/50: jaundice till death on 8/12/50	17/11/50 (1)	7–8/12/50: sulphadimidine 6 g.
4	25/10/47 partial gastrectomy	26/10/47: stomal obstruction—moderate 2/11/47: wound purulent 7/11/47: drainage of pelvic abscess 11/11/47: drainage of perigastric abscess 14/11/47: jejuno-jejunostomy for obstruction 24/11/47: wound fistulae, gastric and faecal 21/12/47: drainage right subphrenic abscess 4/1/48: Division of strangulating band	30/10/47: jaundice, fading after 17/11/47; conjunctival staining until 2/12/47. 26/2/48: jaundice recurred till death on 28/2/48	25/10/47, plasma (2) 30/10/47 (1) 11/11/47 (2), plasma (1) 14/11/47 (1) 4/1/48 (2), plasma (1)	5–7/11/47: sulphadimidine 14 g. 7–13/11/47: soluthiazole I.M. 35 g. 17–22/12/47: sulphadimidine 38 g. 11–16/1/48: sulphadimidine 30 g. 27/4/51–1/5/51: phthalylsulphacetamide 42 g.
5	2/5/51: abdomino-perineal excision of rectum	3/5/51: vesico-perineal fistula 6/5/51: abdominal distension	8/5/51: jaundice till death on 12/5/51	2/5/51 (3)	
6	28/9/50: division of multiple intestinal adhesions	29/9/50: abdominal pain and distension 7/10/50: wound oozing pus 16/10/50: ileostomy for obstruction. Three faecal fistulae developed in wound	3/10/50: jaundice till 29/10/50	16/10/50 (1) 29/10/50 (2)	

* Unless otherwise stated, transfusions were of stored blood; figure following date gives the number of pints.
† Sulphonamide dosage is shown as a total for each course.

TABLE III.—*Details of Liver-function Tests*

Case	Date	Serum Bilirubin mg./100 ml.			Thymol		S.C.G.	S.A.P.	S. Chol.
		Total	Direct	Indirect	T	F			
1	21/2/50	17·6	13·2	4·4	0	Neg.	Neg.	12	157
3	1/12/50	0·7	0·3	0·4					
4	31/10/47	2·9	1·6	1·3	1	,,	,,	3	
	2/11/47	10·0	6·7	3·3	2				
5	8/5/51	2·9	2·0	0·9					
6	5/10/50	8·4	5·5	2·9	1	,,	,,	5	118
	24/10/50				1	,,	,,		
	Sept., 1951	0·6	0·0	0·6	3	1+	1+	8	

T = turbidity. F = flocculation. S.C.G. = serum colloidal gold. S.A.P. = serum alkaline phosphatase, King-Armstrong units. S. Chol. = serum cholesterol mg. per 100 ml.

TABLE IV.—*Histological Findings in Six Cases with Jaundice and in Two Cases in which Death was Due to Peritonitis but which were not Jaundiced*

Case	Abdominal Findings	Liver	Histology of the Liver
1	Diffuse peritonitis; localized pus; r. subphrenic abscess. Leak from duodenal stump	Pale, soft, flabby. Wt. 78oz. (2,210 g.)	Bile-staining in centre of lobule. General vacuolation of liver cells, slightly more marked peripherally. Round-cell infiltration of the portal tracts. Subcapsular zones of necrosis and early fibrosis and congestion, with layer of pus on capsule (see illustration)
2	Fibrino-purulent exudate throughout abdomen. L. subphrenic abscess. Jejunal anastomotic leak	Pale, soft. Normal size	No histology
3	Recent peritonitis: pockets of pus. Fistula from anastomosis of colon	Large, pale, soft. Wt. 69oz. (1,956 g.)	No histology
4	No evidence of peritonitis. All stomata healthy and healed	Flabby. Capsule wrinkled. Wt. 50oz. (1,417 g.)	Acute zonal (centrilobular) necrosis, only small groups of surviving cells. Areas of haemorrhagic exudation and little cellular infiltration. Layer of organized pus on capsule
5	Widespread peritonitis. Large abscess in abdominal wall under suture line. Large infected haematoma round colostomy opening	Fatty	Cells mainly normal; some P.M. change: round-cell infiltration of portal tracts. Recent (probably terminal) thrombosis of portal vein. Layer of superficial pus on capsule
7	Extensive localized peritonitis, with subphrenic and r. perinephric abscess		Superficial layer of necrosis and early fibrosis beneath capsule with sinuses intensely congested below that. Brisk leucocytic infiltration round portal tracts. Scattered foci of fatty infiltration with no particular lobular distribution
8	Several pints of faecal fluid in abdominal cavity due to perforation of duodenal stump following internal herniation and obstruction of afferent loop	Fatty degeneration	Diffuse fatty degeneration affecting the whole of the lobule. Slight round-cell infiltration of portal tracts. No superficial pus. No superficial necrosis

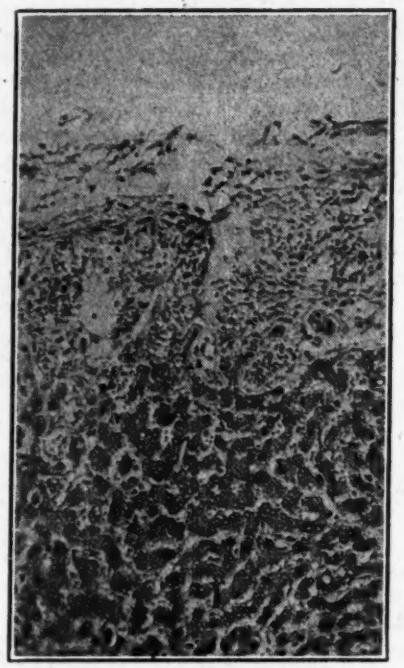
ately bacteriological investigations were made in only three cases, negative swabs being obtained from one, and *Staphylococcus aureus* and *Bacillus aerogenes* from the others.

The anaesthetic agents were those in general use, mainly thiopentone for induction and cyclopropane for maintenance. Chloroform was not used.

Of the six cases four became jaundiced on the fourth and fifth post-operative days, one on the ninth day, and one on the seventeenth. In four cases death occurred from four to seven days after the onset of jaundice and from seven to twenty-one days after operation. Two cases recovered: Case 6, in which jaundice appeared on the fifth day, was lessening on the twelfth day, and had cleared by the twenty-sixth; and Case 4, in which jaundice appeared on the fifth day and remained obvious for twenty days, with conjunctival staining for a further fifteen days. Two months later death rapidly occurred from homologous serum hepatitis, with recurrence of jaundice. Biliuria accompanied the jaundice in all cases.

All cases were febrile at the onset of jaundice, and in the fatal cases fever persisted till death occurred. In the two cases in which jaundice cleared, the fever settled at the same time, or soon after.

It was difficult, in such ill patients, to assess the part played by the liver in the picture, but there was no obvious sudden worsening of the general condition that could be associated with the development of jaundice. Haemorrhagic phenomena were not noted, and bleeding at further operations was not abnormal. Pruritus and bradycardia did not occur, nor was hyperpyrexia found.



Case 1.—Peritoneal exudate, with underlying necrosis of liver cells and fibrosis. (Haematoxylin–eosin. ×100.)

Adequate causes for death, without invoking liver damage, were present in the fatal cases, sepsis being responsible.

All except one patient had received blood transfusions before jaundice appeared, one also having had plasma; no immediate reactions had occurred. Because of the time relationships, these transfusions are not thought to have played any part in causing this type of jaundice. The second and fatal bout of jaundice in Case 4 was almost certainly due to homologous serum hepatitis.

Sulphonamides could not be held responsible, since three patients had them only after jaundice was established, when they gave rise to no apparent adverse effects, one had only a relatively non-absorbable compound, and one did not have sulphonamides at all.

Shock, haemorrhage, or haematoma formation, anaemia, and thrombo-embolism were not thought to have played any part in causing jaundice since they occurred only infrequently.

Table III gives details of the liver function tests carried out. No pre-operative tests were performed. In Case 3 serum bilirubin estimations were carried out three days before jaundice was definitely noted, but unfortunately further estimations were not made. In Case 2 no tests were made, and in Case 5 no tests were made during the second and fatal bout of jaundice.

Apart from Case 3, all the serum bilirubin estimations were raised, with the direct reacting type two to three times as high as the indirect. Flocculation tests were performed four times in three cases (Nos. 1, 4, and 6), and were uniformly negative; the serum alkaline phosphatase was normal in Cases 1 and 6. Flocculation tests carried out a year later in the one surviving case (No. 6) gave abnormal results, although there was no clinical evidence of liver damage, and faecal fistulae were still discharging up to within a short time of the tests.

The post-mortem findings showed peritonitis, mostly widespread, and evidence of leakage from suture lines in all fatal cases except Case 4, in which recovery from sepsis had occurred. In all cases the gall-bladder and bile ducts

were examined, and no evidence of obstruction was found, nor were liver abscesses or pylephlebitis present. The histological findings of Cases 1 and 5 showed no gross abnormality and no typical changes, and did not differ from the findings in two similar patients dying from peritonitis after gastrectomy who did not develop jaundice (Table IV, Cases 7 and 8). Case 4 showed severe acute zonal necrosis due to homologous serum hepatitis. An interesting histological finding in Case 1 was a zone of subcapsular necrosis and early fibrosis bounded by a zone of intense congestion of the sinuses, with a layer of organized pus on the capsule; this appearance was also seen in one of the non-jaundiced cases. A similar layer of pus on the capsule was seen in Case 5, without any zone of necrosis or congestion, and also in Case 4, in which severe zonal necrosis was present throughout the liver.

Differential Diagnosis

The causes of post-operative jaundice fall into four main groups. A double infection is one of the possible causes, and the chance association of acute infective hepatitis or cholelithiasis with the operative condition must be considered, although any other disease causing jaundice may be responsible.

Misdiagnosis resulting in operation in the pre-icteric phase of acute infective hepatitis can occur, but is usually easily recognized. Other conditions, more rarely causing jaundice, which may be mistaken for abdominal surgical conditions are typhoid fever, lower lobar pneumonia, and malignant tertian malaria. A haemolytic crisis can also cause confusion. Acute massive hepatic necrosis may be mistaken for a liver abscess, with its high fever, rigors, and leucocytosis, the jaundice appearing a few days or even up to two weeks after the onset.

Jaundice due to various forms of therapy includes that caused by haemolysis after transfusion, homologous serum hepatitis, and sulphonamide jaundice. Many other drugs have occasionally caused jaundice, but confusion should not arise if that possibility is considered; and this applies also to industrial compounds to which the patient may have been exposed. Jaundice due to haemolysis after transfusion when due to incompatibility is usually easy to recognize. Another type, due mainly to normal destruction of transfused erythrocytes and partly to impairment of hepatic function following operation and anaesthesia, has been described by Geller and Tagnon (1950). They showed that a significant rise in serum bilirubin occurred in 24 of 33 patients undergoing major abdominal surgery with blood transfusion during operation. Jaundice occurred in six, developing before the fourth day, and clearing in a few days. The amount of blood given bore a direct relationship to the rise in serum bilirubin, but not to the appearance or intensity of the jaundice. Ross *et al.* (1947) stated that up to 30% of the erythrocytes stored in acid citrate–dextrose may be destroyed in the first 24 hours after transfusion, depending on the length of time of storage.

Homologous serum hepatitis, unless due to pre-operative treatment, should not present any difficulty in diagnosis, since the long incubation period of 50 to 150 days ensures that it develops during or after convalescence.

Sulphonamides, especially intraperitoneal sulphanilamide, are known to cause jaundice occasionally, although the newer relatively insoluble preparations have not been incriminated in this respect. Jaundice may be due to a hepatitis or to an acute haemolytic anaemia, and the two types may coexist. The incidence of this complication varies considerably in different reports (Jackson and Coller, 1942; Hudson and Smith, 1942; Mueller and Thompson, 1942). Exfoliative dermatitis may accompany the jaundice (Garvin 1938; Long *et al.*, 1940). Spring and Bernstein (1940) reported two cases with coexistent toxic hepatitis and acute haemolytic anaemia, with renal damage, due to sulphanilamide therapy; and Cline (1938) and Russell (1940) each described a case of massive hepatic necrosis after treatment with the same compound. Lederer and Rosenblatt (1942) reported four deaths during sulphathiazole therapy, none showing clinical jaundice, but all showing areas of focal necrosis in all viscera, except the central nervous system. Merkel and Crawford (1942) reported similar findings. The cause of the hepatitis in these patients is not clear. Previous liver disease does not appear to be a factor. Rammelkamp (1948) stated that sulphonamides can be given with impunity to patients with liver disease and jaundice, provided that the drug is not the cause of the jaundice.

Jaundice due to post-operative complications includes that due to absorbed blood pigments from a large haematoma or pulmonary infarct, haemolysis due to septicaemia, surgical operation on the bile ducts, septic jaundice, and acute massive hepatic necrosis. Rarely, haemolysis due to septicaemia can give rise to jaundice. In such cases the indirect reacting type of bilirubin exceeds the direct type, and biliuria does not occur. An obstructive type of jaundice, in the absence of overlooked gallstones, can be due to division of the duct, or to obstruction by ligature, oedema, adhesions, or scarring around the duct or in the region of the ampulla. Such cases, in the absence of sepsis, are usually in fairly good general condition, with negative flocculation reactions, a raised serum alkaline phosphatase, and a raised serum bilirubin, mainly of the direct type.

Acute massive hepatic necrosis occasionally occurs as a post-operative complication. Much of the clinical evidence is likely to be obscured if the patient should also be suffering from peritonitis, and this applies especially to the diminishing size of the liver, the ascites, and the enlarging spleen. The mental state also may not be dissimilar in the two conditions. Unexplained oedema and oliguria, haemorrhagic phenomena, neurological signs, and amino-acidaemia and tyrosinuria are valuable aids in diagnosis. The jaundice can be very deep, and the blood sugar, cholesterol, and prothrombin levels are all low.

Discussion

Study of these cases suggests that a well-defined type of jaundice occurs as a complication of peritonitis. The age and sex incidence is as yet uncertain. The jaundice was moderate or severe, accompanied by biliuria, developing with or soon after the onset of peritonitis, and clearing when the sepsis was overcome or persisting until death. Fever and toxaemia were present at the onset, and persisted with the jaundice. All of these cases followed gastro-intestinal operations, and intraperitoneal leakage of gut contents seemed to be the commonest cause of the complication. There was no apparent deterioration in these ill patients when jaundice appeared, nor did it give rise to fresh symptoms; and evidence of hepatic parenchymal failure, such as oedema, haemorrhagic manifestations, and mental and neurological disturbances, did not occur. The fever and toxaemia, and the frequent fatal outcome, were adequately accounted for by the severe degree of sepsis present. Liver-function tests showed that most of the serum bilirubin was of the direct reacting type, and that the flocculation tests were uniformly negative. The serum alkaline phosphatase levels were normal. Bacteriological investigation was scanty, and organisms were recovered from only two cases, being *Staphylococcus aureus* and *Bacillus aerogenes*. The macroscopic appearances of the liver at necropsy suggested only fatty change without any evidence of biliary obstruction or gall-bladder disease, or of liver abscess or pylephlebitis. Histological examination was carried out in only two cases; the appearances were non-specific, being remarkable perhaps only for the paucity of abnormal findings, and did not differ from the findings in cases of peritonitis of similar aetiology in which there was no jaundice.

The importance of this type of jaundice, as indeed of all other types, depends on the cause. It is a physical sign of peritonitis, and occasionally it may be an early sign. Recognition will also prevent needless and useless exploration of the gall-bladder and bile ducts.

The mechanism of the production can at the moment only be surmised. It seems probable that bacterial toxins, or the organisms themselves, are carried by the portal vein to the liver, giving rise to a hepatitis. Ottenberg and Spiegel (1943) stated that jaundice was an occasional symptom in acute septicaemias, and was usually of unfavourable prognostic significance, with the pneumococcus, haemolytic streptococcus, anaerobic staphylococcus, and *Clostridium welchii* as the commoner organisms responsible. Haemolysis and the effect of bacterial toxins were both usually responsible. Those authors thought that the histological changes in the liver bore no relation to the degree of jaundice. On purely physical grounds peritoneal sepsis would be expected to cause jaundice more often than sepsis elsewhere, owing to the direct drainage of the septic area, often of great extent, direct into the liver.

Another possibility is that the associated ileus and circulatory disturbance give rise to abnormal and toxic gut contents able to affect the liver. Other factors which must be considered are a diminished oxygen content in the portal blood due to stasis, because of the associated ileus, anaemia, and shock, all leading to anoxaemia of the liver. Neither anaemia nor shock played any part in these cases.

The incidence of septic jaundice, compared with that of peritonitis, suggests that the resistance of the liver to the effects of sepsis is high. Although in most of the cases in this series the extent of the sepsis was considerable, equally severe cases of similar duration have not developed jaundice. In the absence of proof to the contrary, however, it seems reasonable to assume that, all other factors being equal, the greater the amount of sepsis the more likely is the development of jaundice. The effects of different organisms in causing it remain to be investigated. The duration of sepsis does not seem to be a factor, since most of these cases occurred early in the post-operative period.

The pre-operative state of the liver may be of importance, but in these cases there was no clinical evidence preoperatively or pathological evidence at necropsy to suggest any predisposing disease.

Summary

Six cases of jaundice occurring after gastro-intestinal operations complicated by peritonitis are described. They form a group for which the name septic jaundice is proposed.

The onset of jaundice was early, usually within the first week of operation, and it persisted until death or until the infection subsided. Signs of hepatic parenchymal failure were not observed. The thymol flocculation and turbidity tests and the serum colloidal gold reaction were consistently negative, and the serum alkaline phosphatase was normal. Treatment and prognosis are those of the peritonitis.

The post-mortem appearances and the liver histology are described; two non-jaundiced cases of fatal peritonitis are included in this section. In some of the cases the liver showed a zone of subcapsular necrosis bounded by a zone of intense congestion of the sinuses, and a layer of pus on the capsule.

The differential diagnosis and the possible factors affecting the liver in this condition are discussed. Severe peritoneal sepsis is thought to be the cause of the condition.

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VITAMIN K IN THE TREATMENT OF ACUTE HEPATITIS

BY

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Since Dam (1935) described vitamin K and Dam and Schönheyder (1936) defined the part it plays in the maintenance of a normal prothrombin level in the blood numerous papers have been published on vitamin K and prothrombin in animals and man. Since 1935 many authors (Hult, 1939–41, among others) have investigated the effect of vitamin K on the prothrombin level in acute and chronic diseases of the liver; and acute hepatitis has also been a subject for research. It was observed that vitamin K administration in these cases exerted a varying influence on the prothrombin level (Butt, Snell, and Osterberg, 1939; Hult, 1940). Begtrup and From Hansen (1942, 1948) found that patients with parenchymal liver damage mostly showed only a slight increase of the prothrombin level after administration of vitamin K. Patients with obstructive jaundice, on the other hand, responded well to the vitamin.

There are several causes of a low prothrombin level. Severe parenchymal liver damage may retard prothrombin formation by the liver, or the absence of bile salts in the intestinal tract in cases with an impeded bile flow may result in a deficient absorption of vitamin K, necessary in some way for the production of prothrombin. The relative extent to which a deficiency in prothrombin formation is due either to liver damage or to defective absorption of vitamin K remains to be determined.

Present Investigation

From January to the beginning of August, 1950, every second case admitted to hospital for suspected hepatitis was on the day after admission put on vitamin K treatment,* one tablet (0.01 g.) three times a day by mouth. The other cases served as controls and were given a placebo.

The material consisted of 247 cases: 178 of acute hepatitis; 5 of subacute hepatitis; 7 of chronic hepatitis or cirrhosis of the liver; 27 of cholecystitis or cholelithiasis (15 stayed two weeks or more in hospital); 13 of carcinoma of the pancreas or the papilla of Vater or neoplasms of the extrahepatic bile ducts; and 17 with various non-hepatic diseases.

The two groups of acute hepatitis consisted of 87 cases in the K (vitamin-K) series and 91 in the C (control) series. The distribution in age groups was practically the same. No attempt was made to divide the material into epidemic and

*" K-vimin " (water-soluble, 2-methyl-1,4-naphthoquinone tetrasodium diphosphate). Astra, Södertälje, Sweden.

inoculation hepatitis. The blood and urine of all patients were tested once a week : icterus index (Meulengracht), thymol turbidity, and prothrombin level (Quick) of the blood; and bilirubin (Hammarsten), bile acids (Hay), and urobilin (Schlesinger) in the urine.

As a rule, the patients did not leave the hospital before the icterus index had returned to 10 (Meulengracht). Since, however, some were discharged earlier, the value of 15 (Meulengracht) or less was taken as a limit in judging the effect of the treatment. Only 2 of the 178 patients were discharged with a higher value. A note was made of the highest icterus index and the time in weeks for a return to 15 or less. In the same way the highest thymol reaction value was calculated, as well as the value at the discharge (see Table I). For bile pigments, bile acids, and urobilin

TABLE I.—*Hospital Stay. Icterus Index (Meulengracht) and Thymol Reaction in the Blood*

	K Series (87 Cases)	C Series (91 Cases)
Hospital stay, days	33·4	30·7
Meulengracht:		
Highest value	59·6	52·7
Increase after admission (cases)	21·0	19·0
Time in weeks for decrease to 15 or less	3·2	2·6
Thymol:		
Highest value	13·2	13·7
At discharge	8·0	8·9

in the urine the time in weeks till negative tests were obtained was noted (see Table II). It will be seen that no statistically significant differences were obtained between the K cases and the controls.

TABLE II.—*Bilirubin, Bile Acids, and Urobilin in the Urine*

	K Series (87 Cases)	C Series (91 Cases)
Bilirubin (Hammarsten):		
Time in weeks pos.→neg. ..	1·2	1·2
Cases pos. at discharge ..	0	2=2·2
Bile acids (Hay):		
Time in weeks pos.→neg. ..	1·4	1·3
Cases pos. at discharge ..	1=1·1	6=6·6±2·6%
Urobilin (Schlesinger):		
Time in weeks pos.→neg. ..	2·4	2·4
Cases pos. at discharge ..	7=8·1±2·9%	12=13.2±3·6%

The *prothrombin* content is placed in relation to the icterus index. The cases are divided according to the icterus index (Meulengracht less than 40, 40–79, and 80 and over). In cases of a mild or rather fairly severe hepatitis (Meulengracht less than 40 or 40–79) no difference was noted in the prothrombin content of the blood in the C and the K series during the stay in hospital (Fig. 1). However, in cases of severe hepatitis (Meulengracht 80 and over) a difference occurred (Fig. 2). The K series showed an increasing average prothrombin content, in spite of an increasing icterus index, as early as one week after admission. The C series, on the other hand, showed a decreasing average prothrombin con-

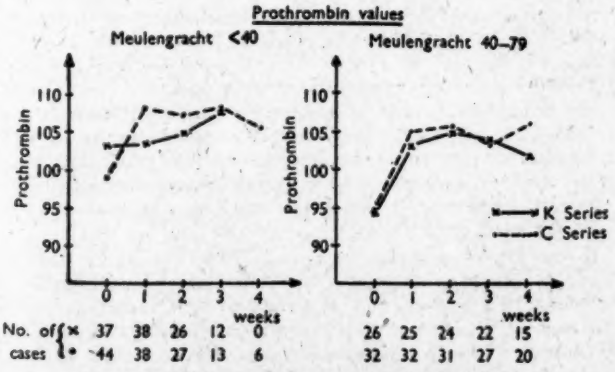

FIG. 1.—Mild and fairly severe acute hepatitis.

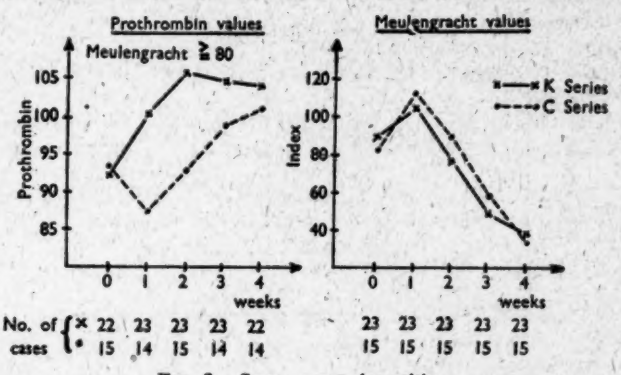

FIG. 2.—Severe acute hepatitis.

tent and increasing icterus index during the first week. Coincidently with a subsequent decrease in the icterus index, the prothrombin content during the following weeks slowly rose to the values of the K series. The greatest difference between the series was in the second and third weeks. The differences—12.6 ± 5.1 and 12.9 ± 5.1, respectively—are statistically significant.

The urinary urobilin test (Schlesinger's reaction) indicates the degree of excretory disturbance in the liver. If no bile, or only an inconsiderable amount, reaches the intestine the urobilin reaction will be negative. In severe hepatitis in both the K and the C series this was noted in about 65% of cases on admission ; therefore the absorption of vitamin K from the intestinal tract must have been considerably impeded. The good response of the blood prothrombin level to water-soluble vitamin K in the K series suggests that, in severe hepatitis, the disturbed absorption of vitamin K is probably the most important cause of the decrease in the prothrombin level. The significance of parenchymal liver damage in these cases is discussed later.

The cases of *obstructive and non-acute hepatic jaundice* are divided into three groups as follows: Group 1, cases of carcinoma of the pancreas or the papilla of Vater, or neoplasms of the extrahepatic bile ducts without metastases in the liver ; Group 2, cases of cholecystitis or cholelithiasis ; and Group 3, cases of chronic hepatitis or cirrhosis of the liver.

Fig. 3 shows the good effect of vitamin K on the blood prothrombin level. The icterus index is roughly the same in both series. The prothrombin level in Groups 2 and 3 is shown in Fig. 4. In both groups the average value was normal on admission—high normal in Group 2 (healthy liver) and a low normal in Group 3 (damaged liver). Neither group showed any effect of vitamin-K treatment. The low normal values in the cases of chronic hepatitis and cirrhosis

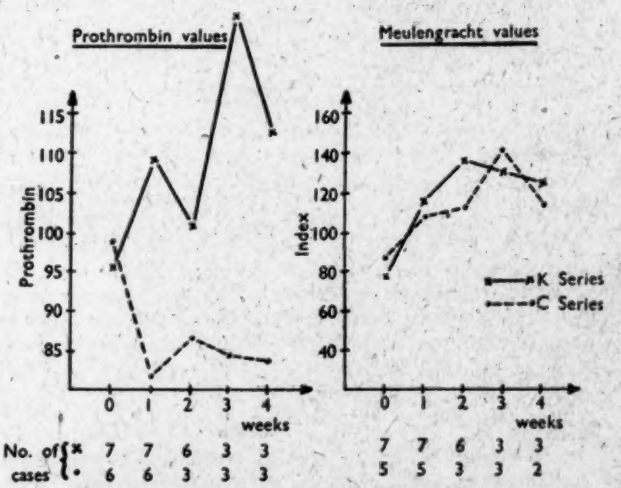

FIG. 3.—Changes in prothrombin content and icterus index in cases of obstructive jaundice (carcinoma of the pancreas or the extrahepatic bile ducts).

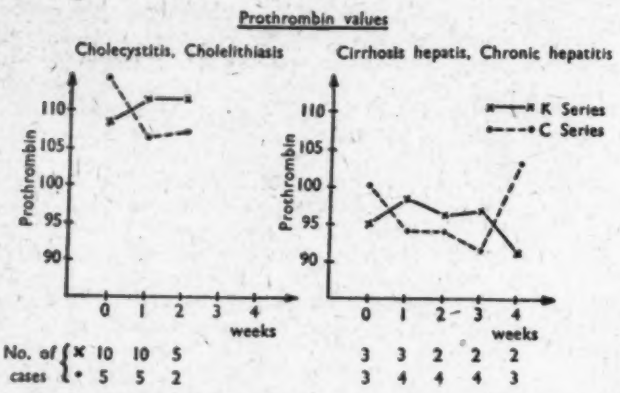

FIG. 4.—Changes in prothrombin content and icterus index in cases of cholecystitis and cholelithiasis (healthy liver) and chronic hepatitis and cirrhosis of the liver (damaged liver).

of the liver may indicate some disturbance in prothrombin formation by the liver. The similar good effects, however, of vitamin K in acute hepatitis and obstructive jaundice show that the parenchymal damage in acute hepatitis affects the prothrombin-producing capacity of the liver only slightly.

Summary

An investigation is made into the effect of oral vitamin K on cases of jaundice, especially those due to acute hepatitis.

Vitamin K did not affect the course of the disease in 178 cases of acute hepatitis. When compared with controls the effect on the blood prothrombin level was obvious only in cases of severe acute hepatitis (Meulengracht 80 and over). In these instances the effect of the oral vitamin was good.

A comparison between treatment of acute hepatitis and of obstructive jaundice failed to show any difference in the effect on the prothrombin value. This indicates that even in cases of severe hepatitis the liver damage does not seriously affect the prothrombin-producing capacity of the liver. Furthermore, it shows above all that decrease of the prothrombin value results from defective vitamin-K absorption in the intestinal tract.

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The annual report for 1951 of the Greenwich and Deptford Hospital Management Committee gives some figures to show the amount of work being done by the typical general hospital. St. Alfege's Hospital, Greenwich, has 504 beds, of which only 366 are staffed and open. In addition, the committee controls the 180 beds of the Miller General Hospital, so there was a total of 684 general beds available, receiving over 10,000 in-patients in the year. The operating-theatres performed nearly 9,000 operations (about 5,000 of them major), and there were 867 live births in the maternity unit. Thus each bed was occupied on the average by 15 patients in the year, for an average stay in hospital of just over three weeks each. Among the work of the special departments may be noted 34,798 *x*-ray examinations, 52,480 blood and other specimens investigated in the pathological laboratories, and 4,142 E.C.G.s taken. Some of this work was, of course, for out-patients. Just under 26,000 new patients were seen here, and there were over 58,000 attendances in the casualty departments.

STRESS INCONTINENCE

A PRELIMINARY REPORT ON A NEW METHOD OF TREATMENT*

BY

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For many years I have been interested in stress incontinence. I have practised most of the methods of repair: anterior colporrhaphy, "buttressing" of the urethra, sling procedures such as Aldridge (1942), Studdiford (1944), Millin I (Millin, 1947) and II (Millin, 1950). So many times has this subject been debated recently and so many different operations have been advised that I almost hesitate to raise the matter again, but my dimming embers of interest were fanned afresh by still another operation, Mulvany (1951). One must pay close attention when a surgeon summarizes his work as follows: "A simple operation, vesico-urethrolysis, for the cure of stress incontinence in women, is described. In a series of 58 cases extending over a period of three years bladder function was restored to normality in all by the operation, and the improvement has since continued unchanged." My immediate reaction to this summary, not an unreasonable one, was one of distrust. I thought that the sooner confirmation or rejection was made, so much the better.

Mulvany's Operation

Mulvany's operation consists in mobilization of the bladder and urethra through a small transverse suprapubic incision and traversing of the retropubic space. The bladder is freed from the back of the symphysis pubis and its lateral supports are also mobilized. Mulvany thinks it likely that the degree of freedom afforded to the sphincter area by manipulations required to prepare the bladder-neck site for the insertion of urethropexy or bladder-raising sutures permits the sphincter muscles to retract and thereby regain normal function. This mobilization of the bladder-neck is a step common to all sling procedures. It is also reasonable to say, although Mulvany does not say so, that while the baby's head is coming through the pelvis the bladder-neck is pressed against the back of the symphysis pubis, and, because of the rich venous plexus there, haematomata may result, giving rise later to adhesions. These are very real arguments in favour of Mulvany's vesico-urethrolysis.

Personal Results

I have performed this mobilization in 23 cases, but have not always used Mulvany's suprapubic approach. In some cases I used the Millin II incision between the external urinary meatus and clitoris to reach the retropubic space. In other cases I have mobilized the bladder off the back of the symphysis through an anterior vaginal wall incision, and then have passed a finger up either side of the urethra through the pudendal diaphragm. I must stress that mine are immediate results, and not long enough has elapsed to assess late cures. I therefore regard this as very much a preliminary report.

Out of 23 cases 21 have been immediately cured. One of the failures was an early case which was done through the Millin II approach and in which a tight band of fibrous tissue, resulting from a previous colporrhaphy, was not cut through. I know now that if I encountered a similar condi-

*Part of an address to the Birmingham and Midland Obstetrical and Gynaecological Society, March 26, 1952.

tion I would use the anterior vaginal wall approach and would cut through that band. A further operation was not done in this case because the woman had secondaries from a carcinoma of the larynx.

The other failure was in a case of moderate cystocele in which mobilization was again done through the Millin II approach. This woman has not yet had the cystocele repaired, but I feel confident that, when it is, the stress will be finally relieved. In this connexion Mulvany (1951) states: "The writer used to confine the suprapubic route to those cases of stress incontinence requiring also an abdominal operation and the vaginal one for those in need of repair, but these excellent results have prompted its employment also in the early to moderate prolapse, especially if there is a likelihood of future child-bearing, as in most cases it is the stress incontinence and not the prolapse which is the most prominent and troublesome feature." I cannot agree with this, and my results show that support from below is as important as mobilization.

Latterly, therefore, nearly all my cases have been done through an anterior vaginal wall incision, whether prolapse was present or not. The procedure when this approach is used consists of: (1) dissection of urethra and bladder-neck; (2) rupture of pudendal diaphragm by index finger or blunt scissors on either side of urethra; (3) pushing the bladder off the back of the symphysis pubis; (4) removal of redundant vaginal mucosa, if any; (5) re-uniting vaginal mucosa and para-urethral tissues. This operation now constitutes part of any prolapse operation when stress incontinence is present.

Many of my cases, but not all, had had several previous operations for prolapse and stress incontinence. One case had also had an Aldridge sling operation by another surgeon, but had not been improved. During operation on that case (suprapubic approach) no evidence could be found of the fascial sling inserted one year before. An immediate post-operative urinary infection tended to obscure the successful result, but one important point made by the patient (and since asked of other patients) was that she could now feel urine passing through the urethra. When the urinary infection was overcome the incontinence was completely relieved.

Conclusions

It would be wrong of me to make statistical deductions with so few cases. But my experience, gained in the study of many other procedures besides the present series, leads me to the following conclusions.

1. That cases of stress incontinence require careful radiographic and cystoscopic investigation, apart from clinical neurological examination, before operations are performed. There is nothing new in this, as has been repeatedly pointed out by Millin (1950) and Winsbury-White (1948). Measured cystography has shown that there is often a slight increase in descent of the bladder-neck after mobilization, although the stress is cured.

2. That Mulvany has introduced a new aetiological concept and an operation which has formed part of many sling procedures of the past.

3. That Mulvany's mobilization is not necessarily a sufficiency in itself. There must be adequate support to the bladder-neck from below. These two factors—mobilization and support from below—can be accomplished through an anterior vaginal approach. There should be no need for sling procedures if this is so.

4. That the mobilization can either be done from above in the absence of prolapse (as an accompaniment of an abdominal operation) or from below during plastic repair operations even when the urethral bulge is minimal.

5. That these cases require careful post-operative assessment of cure. Often the stress is replaced either by frequency because urinary infection is common, or by urgency, particularly when the bladder is full—for example, in the morning. Urethral sensation of flow in some cases accompanies successful cure of stress.

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ICTERUS GRAVIS AND CIRRHOSIS OF THE LIVER

BY

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Braid and Ebbs (1937) reported the case of a child who had been deeply jaundiced in infancy, who subsequently developed coarse multilobular hepatic cirrhosis, and who died at the age of 3 years and 8 months, after a haematemesis. This child, the second and last in the family, became jaundiced on the third day of life. The jaundice persisted for two months, by which time the haemoglobin was 74% and the red blood corpuscles numbered 3,900,000 per c.mm., 5.8% being reticulocytes. At the age of 1 month the liver was firm and hard, and its lower edge reached the level of the umbilicus; the spleen was never felt during the neonatal period, but became palpable at 5 months. At the age of 3 years 8 months the child was readmitted to hospital with ascites; a few days later she had a haematemesis and died.

Drummond and Watkins (1946) have more recently given details of three families, in two of which Rh iso-immunization may have been the cause of hepatosplenomegaly in the children; but in these families the early history was not suggestive of Rh iso-immunization, for the affected children were never noticeably jaundiced or anaemic in infancy. Moreover, the maternal serum in each case contained no detectable Rh antibody; in one case this was only five years after the birth of the last child in the family—this child had died, deeply jaundiced, on the third day of life—and in the other case only two years after the last of three successive stillbirths. My own experience (Gerrard, 1951) suggests that, once iso-immunization has occurred, Rh antibodies persist for at least eight years, and often longer. However, both mothers in the two families mentioned above were eventually sensitized, for each had a rigor and each produced Rh antibodies when transfused with Rh incompatible blood. In Drummond and Watkins's third family the early history, as in Braid's case, was suggestive of Rh iso-immunization, for the affected child became jaundiced within a day or two of birth, and the jaundice persisted for three months; but in this instance Rh iso-immunization was excluded as a causative factor.

Nussey (1949) has also recorded a family in which Rh iso-immunization may have been the cause of hepatosplenomegaly in three siblings, but the early history of this family was not suggestive of Rh iso-immunization, for none of the three children with hepatosplenomegaly had ever been noticeably jaundiced or anaemic in infancy, and, though the children were Rh-positive and the mother Rh-negative, the maternal serum contained no Rh antibodies either in saline or in albumin. This was 17 years after her last pregnancy, by which time she might well have lost her antibodies.

An Earlier Review

I have recently reviewed (Gerrard, 1951) nearly all families from which cases of haemolytic disease of the newborn were admitted to the Children's Hospital, Birmingham, between the years 1927 and 1949 inclusive. The purpose of this review was to elicit the incidence and main sequelae of haemolytic disease of the newborn and to find out if hepatic cirrhosis might be one of these. I set out to trace 321 families; only 10 were finally unaccounted for, and four more had moved too far afield to be visited. The remaining 307 families were visited—often more than once—blood was taken for serological investigation, and a detailed family history compiled. In 81 haemolytic disease had almost certainly not been present. In retrospect it is not very surprising that in the early days quite a number of cases were misdiagnosed. In the other 226 families the family history and serological findings supported a diagnosis of haemolytic disease due to Rh iso-immunization in one or more of the siblings.

The mortality of icterus gravis before 1942 was high (46% of such patients admitted to this hospital between 1927 and 1942 died; during the next seven years only 23% of admissions died); the proportion of older children surviving was therefore small. In addition, before 1942 most infants with icterus gravis died at home; after 1942 there was an increasing tendency for them to be admitted hurriedly to hospital. Latterly the number of such admissions has fallen, for these cases are now nearly always treated in the maternity hospitals in which they are born. In my material 293 out of a total of 381 had survived the neonatal period; all these were re-examined.

Later Investigation

Blood was taken from all children aged 5 or more and from all cases of kernicterus, for in these jaundice tends to be greater and liver damage might therefore be expected to be more frequent. The serum albumin and globulin and the thymol turbidity were estimated in these cases and also in the first group of younger children who were reviewed; I did not, however, persist with this investigation in the younger children, for they all registered a hearty disapproval of this mode of approach and were clinically well. It was decided to estimate the serum albumin and globulin, because Higgins *et al.* (1944) have stated that "disturbances of the plasma proteins are an outstanding feature in subacute and chronic cases of hepatitis, and a persistent alteration of the albumin/globulin ratio indicates that the liver has suffered irreparable damage." The thymol turbidity was also estimated, as it is often raised in chronic hepatitis: according to Maclagan (1947) a thymol turbidity greater than 4 is pathological; in normal people it is rarely greater than 2. The combination of a careful clinical examination with these biochemical findings would, it was hoped, bring to light all cases with a persisting hepatic defect.

My findings in the 79 children submitted to both lines of investigation are recorded in Tables I and II. In Table I are recorded the results of the liver-function tests and other findings in the cases of kernicterus. The thymol turbidity was greater than 2 in Cases 13, 14, 20, and 21; in Cases 14 and 21 there was also a reversal of the albumin/globulin ratio; in one other case (No. 3) the normal albumin/globulin ratio was altered. In none of these cases was the spleen palpable, and in none was the liver either enlarged or unduly firm. In Table II are recorded the results of the liver-function tests and other findings in the cases of uncomplicated icterus gravis. When I examined these children I found the spleen palpable in Cases 51, 60, 67, 77, and 78; in all these cases it was firm, but in no instance was the liver either enlarged or hard. In Case 78, as also in Case 53, the normal albumin/globulin ratio was altered. The thymol turbidity was definitely pathological in Cases 30 and 56, but Case 56 had recently had an attack of infective hepatitis.

TABLE I.—*The Serum Albumin and Globulin and the Thymol Turbidity in Cases of Kernicterus*

Case No.	Age in Years	Albumin (g./100 ml.)	Globulin (g./100 ml.)	Thymol Turbidity (Units)	Duration of Jaundice	Comments
1	1	4·5	2·6	1·0	5 weeks	
2	1	4·4	2·6	2·0	12 ,,	
3	2	2·85	2·81	1·5	4 ,,	Teeth green
4	2	4·48	1·75	1·5	4 ,,	
5	2	4·79	2·30	1·5	4 ,,	
6	3	3·94	2·63	1·5	16 ,,	,, yellow
7	3	3·76	2·25	1·0	16 ,,	
8	3	3·73	2·06	2·0	13 ,,	,, ,,
9	3	5·2	2·1	1·5	4 ,,	
10	4	4·0	2·6	1·0	8 ,,	,, green
11	4	4·2	2·7	2·0	4 ,,	,, yellow
12	4	4·55	1·5	1·5	13 ,,	
13	5	3·10	2·46	2·5	13 ,,	,, ,,
14	6	3·95	4·24	2·5	16 ,,	
15	7	4·5	2·7	1·5	7 ,,	
16	8	3·08	2·11	1·0	2 days	
17	9	4·81	2·65	2·0	3 weeks	,, green
18	10	4·6	2·73	1·5	16 ,,	
19	10	4·54	1·65	1·0	6 ,,	,, ,,
20	10	4·34	2·79	3·0	12 ,,	,, ,,
21	12	3·38	4·12	2·5	1 week	
22	15	3·77	2·45	1·0	6 weeks	,, ,,
23	16	4·51	2·05	1·0	3 ,,	
24	18	3·79	1·95	2·0	1 week	
25	20	4·62	2·2	2·0	1 ,,	
26	21	4·6	2·7	1·0	12 weeks	
27	22	5·04	2·67	1·5	2 ,,	

Cases 8, 20, and 22 had a transient obstructive jaundice in infancy. The colour of the teeth refers to the deciduous teeth only.

TABLE II.—*The Serum Albumin and Globulin and the Thymol Turbidity in Late Cases of Icterus Gravis*

Case No.	Age in Years	Albumin (g./100 ml.)	Globulin (g./100 ml.)	Thymol Turbidity (Units)	Duration of Jaundice	Comments
28	2	4·33	2·75	2·5	1 week	
29	3	4·03	2·88	2·5	2 weeks	
30	3	3·7	2·2	6·5	8 ,,	Teeth green
31	3	3·8	2·1	2·5	2 ,,	
32	3	4·7	2·3	1	1 week	
33	3	3·61	2·29	1·5	None	
34	4	4·21	2·03	2·5	,,	
35	4	3·3	2·0	1	6 weeks	
36	4	4·23	1·45	1	1 week	
37	5	3·2	1·8	1	1 ,,	
38	5	3·9	2·4	2·0	10 weeks	
39	5	4·1	2·4	1·5	12 ,,	
40	5	4·2	2·4	2·5	3 ,,	
41	5	4·1	1·9	1·5	2 ,,	
42	5	4·6	2·1	1·5	4 ,,	
43	5	4·6	2·1	1·5	12 ,,	Teeth green
44	5	3·76	3·42	2·5	8 ,,	
45	6	4·91	2·74	2·5	4 ,,	
46	6	3·7	2·9	2·0	1½ ,,	
47	6	4·6	2·1	1·5	6 ,,	
48	6	3·9	1·6	1·5	None—only anaemia	
49	6	4·1	2·3	1·5	3 weeks	
50	6	3·9	2·4	2·0	3 ,,	Deaf
51	6	3·8	1·6	1·5	6 ,,	Spleen tipped
52	6	4·07	2·72	1	2 ,,	
53	6	3·6	4·02	1	14 ,,	
54	7	3·7	2·0	2·5	8 ,,	Teeth yellow
55	7	3·55	1·96	2·5	4 ,,	
56	7	3·8	2·3	4	3 ,,	Infective hepatitis 6 months previously
57	7	3·49	1·77	1·5	8 ,,	
58	7	4·19	3·17	0·5	11 ,,	Teeth green
59	7	4·56	2·63	1	14 ,,	,, ,,
60	8	4·09	1·8	1	2 ,,	Spleen tipped
61	8	4·22	3·82	0·5	4 ,,	
62	8	5·0	2·7	1·5	3 ,,	
63	8	3·33	2·96	0·5	2 ,,	
64	8	4·12	2·46	1	2 ,,	
65	9	3·65	3·23	1·5	4 ,,	
66	9	3·59	1·89	1	2 ,,	
67	9	3·79	1·86	2	1 week	Spleen tipped
68	9	4·03	3·67	2	3 weeks	
69	10	4·39	3·34	1·0	10 days	
70	10	3·31	1·19	2·5	4 weeks	
71	10	4·66	1·66	1·5	10 ,,	
72	13	3·8	2·7	<1	3 ,,	
73	14	4·58	1·63	1	1 week	
74	14	5·2	1·41	1	6 weeks	
75	14	3·81	2·70	<1	12 ,,	
76	15	4·52	1·46	<1	2 ,,	
77	15	3·2	2·8	1	14 ,,	Teeth green; spleen tipped
78	15	3·2	3·5	<1	12 ,,	Spleen tipped
79	17	4·7	2·44	1	2 ,,	

Case 57 had a transient obstructive jaundice in infancy. The colour of the teeth refers to the deciduous teeth only.

The above findings suggest that Rh iso-immunization may result in some degree of hepatic insufficiency, for in 11 of the 79 cases (including the child who had recently had an attack of infective hepatitis) of which details are given there was clinical enlargement of the spleen, or a reversal of the albumin/globulin ratio, or an abnormal elevation of the thymol turbidity. The findings, however, do not suggest that Rh iso-immunization is commonly followed, in childhood, by hepatosplenomegaly.

It has been suggested that familial hepatic cirrhosis, although an uncommon condition, is related to Rh iso-immunization; I therefore took a full and careful family history in each of the 226 families reviewed, which included these and other cases; in no instance was there any evidence that siblings of children who had had haemolytic disease of the newborn had either developed or had died of cirrhosis of the liver, but I did not examine as a routine children who had not been jaundiced or anaemic in infancy.

Discussion

Nearly all infants with haemolytic disease of the newborn who live for more than a few hours show a variable degree of jaundice. This jaundice often deepens rapidly and may even become obstructive in character; the stools become colourless and the urine dark, and the liver and spleen are usually large and firm. If the infant dies at this stage the liver is found at necropsy to be large and green and its surface finely granular; on microscopy an early biliary cirrhosis is evident (Hawksley and Lightwood, 1934; Baar, 1946; Zollinger, 1946). Death, in our experience, has usually been associated with an intercurrent infection—for example, gastro-enteritis—but this was not so in two cases recorded by Lightwood and Bodian (1946) in which the infants developed ascites and died at 7 and 10 weeks of age; a third died at the age of 6 months, but with an associated otitis media and gastro-enteritis.

More commonly the infant survives the obstructive phase; the stools again become pigmented, the jaundice gradually fades, and the hepatic and splenic enlargements subside. At the Children's Hospital, Birmingham, we have not encountered any cases as severe as Lightwood and Bodian's Case 1, which developed ascites and required abdominal paracentesis on two occasions before making a spontaneous recovery; but we have had six cases which survived the obstructive jaundice and are now well. None required operative intervention. But if an obstruction occurs—for example, in the common bile duct—and is due to pigment stones, as in Lightwood and Bodian's Case 3, or to inspissated bile, as in Case "Hi" recorded by Skelton and Tovey (1945), this may be relieved surgically. Two cases of haemolytic disease of the newborn associated with obliteration of the common bile ducts have also been recorded (Pasachoff, 1935; Skelton and Tovey, 1945), but no comparable case has been seen at this hospital.

Biliary cirrhosis associated with haemolytic disease of the newborn is by no means rare (Zollinger, 1946; Craig, 1950), but the immediate prognosis, in our experience, seems to be good. In order to determine the more remote prognosis and also to determine whether any light can be shed on the aetiology of familial hepatic cirrhosis, this review of children who had had icterus gravis was undertaken.

A number of cases of familial hepatic cirrhosis have been reported (Parkes Weber, 1903; Bramwell, 1910; Gunn, 1926; Poynton and Wyllie, 1926; Langmead, 1934; Bateman, 1934; Debré, 1939). If the cases recorded by Drummond and Watkins (1946) and by Nussey (1949) are included, only 3 of the 29 cases were jaundiced in the neonatal period; this does not suggest that icterus gravis is a precursor of familial hepatic cirrhosis.

F. Braid (personal communication) has recently had under her care at this hospital two children, a girl and her brother, with hepatosplenomegaly. The mother is O Rh-positive, genotype R_1R_2 CDe/cDE; her serum contains no anomalous agglutinins or blocking antibodies. The father is O Rh-negative, genotype rr cde/cde. The first child in the family, a boy, was born in 1947; he became jaundiced when he was 1 month old and the jaundice persisted for four and a half months. He remained in good health until he was 1 year old; he then developed ascites; he died of a coarse nodular cirrhosis at the age of 2½ years; his blood group was O Rh-positive, genotype R_2r cDE/cde. The second child in the family, born in 1948, had never been jaundiced or anaemic, his liver was palpable but not enlarged, there was no splenomegaly; his blood group was O Rh-positive, genotype R_1r CDe/cde. The third child in the family, aged 1 year, became jaundiced when he was 5 days old, the jaundice persisted for three months and was associated with the passage of pale stools; he had a large hard liver and spleen; his blood group was O Rh-positive, genotype R_1r CDe/cde. The indirect Coombs test, using the maternal serum and the erythrocytes of the youngest child, was negative. The family history in this case was suggestive of Rh iso-immunization, but the investigations revealed that Rh iso-immunization could not be the cause of the hepatosplenomegaly.

My own findings and a critical review of the literature do not suggest that Rh iso-immunization often, if ever, gives rise to hepatic cirrhosis later in life, and even when the child has been deeply jaundiced in infancy there is surprisingly little clinical evidence, in my material, of severe liver disease later in childhood. The only really suggestive case is that reported by Braid and Ebbs (1937), and, though the mother of this child was Rh-negative and her husband Rh-positive, her serum contained no detectable Rh antibodies, though this was 15 years after the birth of the affected child. It would appear, then, that hepatosplenomegaly as a late sequel of Rh iso-immunization is rare. If it is as unusual as my findings suggest, its occurrence in more than one member of a family would not be anticipated unless there was an underlying constitutional defect. Parkes Weber (1947) suggests that familial hepatic cirrhosis is analogous to other familial conditions associated with cirrhosis of the liver—for example, hepato-lenticular degeneration—and that it is, as he puts it, a congenital developmental defect. I do not think that my findings permit of a more detailed explanation. It would appear that familial hepatic cirrhosis, like hepato-lenticular degeneration, is not directly related to Rh iso-immunization, but it is possible that Rh iso-immunization may occasionally bring to light or accentuate an underlying latent hepatic defect.

Summary

Seventy-nine children who had had haemolytic disease of the newborn have been re-examined. In 11 instances evidence was obtained suggesting some degree of liver dysfunction, but no cases of frank hepatic cirrhosis were encountered, nor was any evidence collected which might support the thesis that familial hepatic cirrhosis is a sequel of Rh iso-immunization.

I am grateful to Dr. H. S. Baar and Dr. F. Braid for their help and advice, to Dr. E. M. Hickmans for undertaking the biochemical investigations, and to the staff of the Children's Hospital, Birmingham, for giving me access to their cases.

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EFFECT OF LIVER SUPPLEMENT ON GROWTH OF CHILDREN

BY

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In the course of work on dietary reproductive factors in the rat (Wiesner and Yudkin, 1951) it was found, though not then reported, that supplements of liver caused a small but significant improvement in growth even when the animals were fed an apparently complete stock diet consisting of non-purified foods (Fig. 1). The effect was produced both by fresh mammalian liver and by a specially prepared liver powder; other liver powders did not produce this promotion of growth. It was decided to test whether a similar effect on growth could be produced in children.

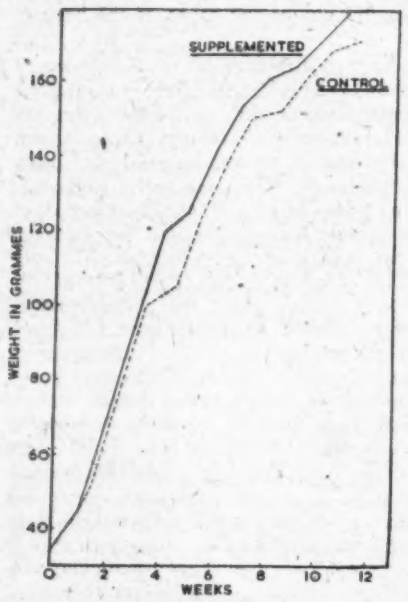

FIG. 1.—Effect of liver supplement on growth of rats. Each group contained 12 female rats of strain KC$_1$, housed individually. From weaning, rats fed *ad libitum* on stock diet (see Wiesner and Yudkin, 1951). Supplement consisted of 1 g. of fresh liver daily to each rat in the "liver" group.

Experimental

Beef liver was dried in a special way under careful supervision. After establishing that it was active in promoting growth in rats, it was incorporated into bars of chocolate with an orange flavour, so that each bar of about ¼ oz. (20 g.) contained 2 g. of liver powder (L). Bars of chocolate of the same size and flavour but of different shape and containing no liver (C) were also made for administration to children acting as controls.

The children studied were from four day nurseries in West London. All children over 2 years and under 3 years took part in the experiment; these numbered 75, of whom 40 were boys and 35 girls. They were all apparently well, and the average height and weight were up to standard, being, for example, slightly though not significantly greater than the figures for London children recorded by Gore and Palmer (1949). On the basis of their age, height, and weight at the beginning of the test, they were divided into two matched groups, so that about an equal number of both boys and girls at each school, of about the same age, height, and weight, fell into each group. The matron in each school was then instructed to give the children in one group chocolate "L" and those in the other group chocolate "C"; each child received one bar a day during the five days of attendance each week, so that the children in the experimental group took 2 g. of dried liver on each school day, equivalent to 12 g. of fresh liver. The arrangements for distributing the chocolate, either at one time or in divided amounts, differed in the different nurseries, but there was no doubt that every care was taken to ensure that each child received the type of chocolate allotted to him. The appetite of the children was apparently not affected by the chocolate.

Measurements of height to the nearest ¼ in. and weight to the nearest ¼ lb. were made by me with checked measuring-rods and weighing machines (¼ in. = 0.6 cm.; ¼ lb. = 113 g.). Each child, without clothes, was measured once 10 days before supplementation, once three days before supplementation, twice more during the next 10 weeks, and finally 13 weeks after the beginning of supplementation. These repeated measurements made it possible to eliminate gross errors in recording and to minimize errors in measurements. The children were examined at random, and the names and measurements recorded by a personal assistant. The group to which each child belonged was entered in the record only after the final examination.

Results

Of the 75 children with whom the experiment began, 15 left during the test, so that records of growth were available for 60 children—31 boys and 29 girls. No differences in growth between the boys and girls were evident, so that the results for both sexes have been combined.

Of the 60 children, 28 (14 boys and 14 girls) were in the control group and 32 (17 boys and 15 girls) in the experimental group. Because some of the children had left, the initial age, weight, and height of the control children were now on average slightly greater than those in the experimental group (Table I). The differences in height and weight were just about those expected from the difference of nearly two months in age, although statistically the difference in age, height, or weight between the two groups was not significant. The average absences from the nurseries were almost the same for the two groups. From the absences it can be calculated that the average consumption of liver powder by the children in the experimental group amounted to 1.2 g. daily for the 91 days (13 weeks) of the test.

TABLE I.—*Comparison of Control and Experimental Groups. Figures Give Mean Values and Standard Errors.*

Group	No.	Initial Age (Months)	Initial Height (in.)	Initial Weight (lb.)	Absence Days
Control	28	31·2 (±0·63)	35·8 (±0·30)	30·7 (±0·57)	12·6 (±2·08)
Experimental ..	32	29·4 (±0·59)	35·3 (±0·25)	29·9 (±0·55)	13·3 (±1·98)

Differences between groups not significant ($P > 0.05$).

After 13 weeks of supplementation appreciable and significant differences in growth were found in the two groups (Table II, Fig. 2). Compared with the control children, the children receiving the liver chocolate gained 0.22 in. (0.55 cm.) or over 20% more in height, and 0.63 lb. (285 g.) or nearly 40% more in weight. That these results were statistically highly significant, with a probability of 1 in 100 or more against their being due to chance, was shown both by analysis of variance of the individual gains in height or weight and by the χ^2 test applied to the number which gained more or less than the average for both groups. The effect of the liver was apparently not related to the original "nutritional status" or physique of the children,

TABLE II.—*Effect of Liver Supplement on Growth*

Group	No.	Increase in Height (in.)	Increase in Weight (lb.)	No. (and %) with Gain above Average	
				Height	Weight
Control	28	0·98 (±0·049)	1·64 (±0·017)	11 (40)	10 (35)
Experimental ..	32	1·20 (±0·050)	2·27 (±0·016)	23 (70)	24 (75)

Differences between groups significant ($P = 0.01$ or less).

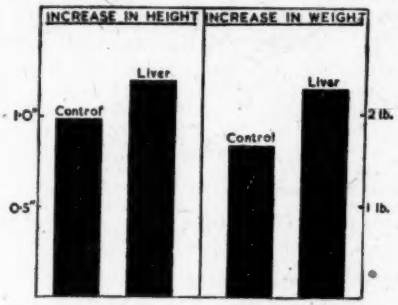

FIG. 2.—Effect of liver supplement on growth of children.

for very similar gains occurred in children with either a high or a low Tuxford index (Tuxford, 1942).

Discussion

The effect of the liver preparation on growth has thus been shown to occur in children as well as in rats. In neither instance was there any reason to suppose that the unsupplemented diets were in any way deficient. It has already been mentioned that the children were apparently well and healthy.

This work recalls that of Wetzel, Fargo, Smith, and Helikson (1949) and of Chow (1951), in which oral vitamin B_{12} was shown to promote the growth of children. Wetzel *et al.* gave 10 μg. daily to 11 malnourished boys and girls aged between 5 and 12 years and found that five of them showed a significant improvement in physique as measured by their progress along a "Wetzel grid." Chow studied two groups of children, the first of 45 chronically ill or malnourished children from 6 months to 12 years, and the second of 18 normal children from 18 months to 4 years. About half of the children in each group received 10 or 25 μg. of vitamin B_{12} daily. After three months for the ill or malnourished children, and six months for the normal children, there was a significantly greater gain in weight in those receiving the vitamin.

The work of these authors differs in three respects from that described in this paper. First, their groups were both smaller and much less homogeneous as regards age, weight, and height. Chow himself stresses that "owing to the heterogeneity of the subjects with chronic illness and the small numbers of the normal children studied, the results are merely considered suggestive of trends." Not only was the present study made on 60 children, but the narrow range of age, and the distribution according to sex, height, and weight, ensured that our subjects were more homogeneous and the groups better matched.

Secondly, apart from the small group of 18 normal children studied by Chow, both he and Wetzel *et al.* were dealing with ill and malnourished children. The children of the present study were apparently well, and in particular there seemed to be no relation between improvement in growth and the "nutritional state" of the children as measured, for example, by the Tuxford index.

The third difference between this work and that of the other investigators is in the nature of the supplement. It is of course tempting to suppose that the liver produced its effect because of its content of vitamin B_{12}. However, there are several indications that this was not so. (1) Vitamin B_{12} does not always produce an increase in growth in children, for Downing (1950) has reported that it does not stimulate the growth of premature infants. More recently, Benjamin and Pirrie (1952) have not been able to confirm the results of Wetzel *et al.* and of Chow on chronically ill children. (2) Microbiological assay has shown that our liver powder contained rather less than 1 μg. of vitamin B_{12} per g. Although there is evidence that the availability of the vitamin from liver is not the same for micro-organisms as it is for human beings, it is unlikely that the average amount of 1.2 g. of liver powder daily supplied anything approaching the 10 to 25 μg. given by the American workers. (3) A preparation of fish liver known to be an active source of vitamin B_{12} was not active in promoting the growth of rats on the stock diet. (4) Continued (unpublished) experiments with rats have shown that, unlike liver, vitamin B_{12} added to the stock diet does not produce an increase in growth.

It would seem probable, therefore, that the factor responsible for the effect on growth is not vitamin B_{12}. It should also perhaps be added that the growth factor is likely to prove different from "reproductive factor R," since the preparation of fish liver which did not promote growth was nevertheless a potent source of "factor R" (Wiesner and Yudkin, 1951).

Work is being continued to determine the distribution and nature of the growth factor. Until further information is available it cannot be assumed that the factor is present in all preparations of liver. This report is nevertheless presented at this stage, since it seems sufficiently interesting that liver contains what appears to be a new nutritional factor, which produces such a significant effect on the gain in height and weight of children in such a short time.

Summary

Experiments with rats showed that growth was significantly improved when a powdered preparation of liver was added to a supposedly complete stock diet. A similar preparation was given to apparently normal children of 2 years of age, at the average rate of 1.2 g. a day. After 13 weeks the 32 children taking this supplement gained on average about ¼ in. (0.6 cm.) more in height and 10 oz. (285 g.) more in weight than the 28 control children. These gains were roughly 20% and 40% more than those of the control children. Reasons are given for the belief that the dietary factor responsible for this improved growth was not vitamin B_{12}.

I am indebted to Messrs. Kylon Ltd. for the special preparation of liver powder (Kylon G.F.), to Messrs. Cadbury Bros. Ltd. and their chief chemist, Mr. R. V. Wadsworth, for the manufacture and supply of the chocolate, and to Dr. W. F. J. Cuthbertson, of Glaxo Laboratories, for the assays of vitamin B_{12}. I am especially grateful for the help and co-operation of Sir Allen Daley (lately Medical Officer of Health, L.C.C.), Dr. V. Russell (Divisional Medical Officer, L.C.C.), and the matrons and their colleagues at the four day nurseries.

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H. Kalmus, H. L. Nixon, and C. R. Ribbands read a paper to the Royal Society on June 19 on the origin of the odours by which honeybees distinguish their companions and the division of labour and food transmission within the honeybee community. During her lifetime every honeybee performs a sequence of tasks, the oldest bees being foragers. Most bees omit some of the earlier duties and tasks are allocated in accordance with the needs of the colony. These needs are determined by food supplies and appreciated through food transmission. Widespread food transmission or food sharing was demonstrated by a tracer technique. All the major tasks involve the manipulation of food, and through food transmission all the bees performing any task are evenly fed. Any surplus causes the older members to leave their group and join another. Experiments proved that foraging bees recognize their hivemates by their odour, which they distinguish from the odour of other honeybees. The common colony odour is derived from the food transmission, which provides an identical diet with identical odorous waste products. Food transmission is therefore the most primitive and the most important method of communication of the honeybee community.

TUBERCULOSIS SURVEY IN SCHOOL-CHILDREN IN THE CALDER VALLEY

BY

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Since the turn of the century different investigators have conducted surveys to determine the state of tuberculin sensitivity of different communities and races. Many of these older inquiries were originally of great epidemiological value but have become somewhat dated by the passage of time. Some of the more recent medical literature has assisted in presenting a picture of the changing epidemiological pattern during the past 50 years. Yet criticism has been directed against a number of these publications on the grounds that there has been a somewhat arbitrary selection of material and that the investigated groups are not necessarily representative samples of the community—namely, out-patients at tuberculosis clinics, in-patients in children's or chest hospitals, etc. A further problem in the interpretation of many results has been the striking lack of uniformity in technique and equipment employed by the various workers.

In 1949 the Medical Research Council decided to embark on a large-scale national survey to determine the level of tuberculin sensitivity among the school population in different regions in the country and to couple this with a miniature *x*-ray film of each tuberculin-tested child. The West Riding County Council of Yorkshire expressed its willingness to participate and co-operate with the M.R.C. in this venture.

There appeared to be two methods of approach in organizing this inquiry: the investigation of all scholars from a number of *selected* schools spread over a large region, or from *all* schools in a much smaller area of the county. After some deliberation it was decided to pursue the latter course, and thus an intensive survey of the entire school population of one divisional public health area of the West Riding of Yorkshire was adopted as our local policy. The Calder divisional public health area was selected. This is essentially rural in character but industrial in population. The reason for this apparently contradictory statement is that the area covers over 60,000 acres, with a population of under one per acre. The bulk of the population is concentrated into the deep valleys. Most of the housing is of the back-to-back type, and much of it is actually built into the hillsides. Small factories and mills intermingle with the houses. The principal industry is textiles, with marginal farming on the upland slopes. The largest towns in the area are Sowerby Bridge and Todmorden, with populations of approximately 20,000, and Hebden Royd, with a population of 10,000. A few statistics relating to 1948 will illustrate the general health of the population more adequately than any description.

Birth rate	16·4 per 1,000
Death rate	14·7 per 1,000
Infant mortality	39 per 1,000 live births
Tuberculosis morbidity (all types notified)	460 per 100,000
Tuberculosis morbidity (respiratory notified)	245 per 100,000
Mortality due to pulmonary tuberculosis	30 per 100,000
Mortality due to pulmonary tuberculosis (England and Wales)	44 per 100,000

Method of Investigation

The technique was that prescribed by the M.R.C. Tuberculosis Survey Committee for all areas involved in the national Survey. The interscapular area of each child was cleansed with acetone and then gently stroked six times with a 1-in. (2.5-cm.) wide strip of flourpaper (*British Medical Journal*, 1950; Dick, 1950). A "V" was then written with tuberculin jelly on the abraded zone of the child's back, and this was covered with adhesive plaster. The results were read 48 to 72 hours later by one of us (G. A. W.) to ensure uniformity. Findings were regarded as positive when there was an area of oedema with surrounding erythema at least 5 mm. in diameter at the site of application of the jelly. In subjects who failed to give a positive response a further test (intradermal) with 1 mg. of old tuberculin, 1/100 dilution, was undertaken. Here the standards of interpretation were those adopted by Aronson (1933) and the Prophit Survey Report. Each child had a miniature (35 mm.) film taken, interpreted by one of us (S. K.), and if any abnormality was detected this was followed by a large chest film. All children showing abnormalities on the large skiagram had a full clinical investigation carried out by one of us (B. M.).

TABLE I

	No.
Total school population	6,532 (100%)
Permission received for investigation	5,805 (88·7%)
School population mass-*x*-rayed and tuberculin-tested	5,552 (95·6%)
Defaulters	253

The entire schoolchild population of the Calder Valley area were invited to participate in this survey and 85% were investigated (Table I); this represents 95.6% of the children who expressed their willingness to co-operate in this inquiry. In an area with a total population of 57,454, about 20% of which is grouped in small rural communities of 50 to 1,000, this response must be regarded as extremely satisfactory.

Results

The results following tuberculin-testing according to age groups and without differentiation of sex are shown in Table II and Fig. 1. The sex incidence of positive reactors is shown in Fig. 2.

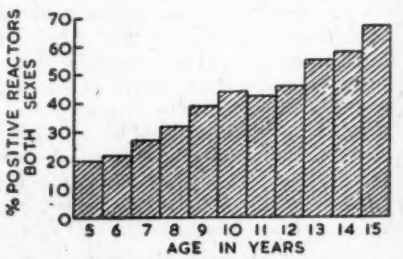

FIG. 1.—Results following tuberculin testing.

Reactor Rate and Location of Schools in the Calder Valley Area.—The 49 schools in this public health area were subdivided into three groups—urban, rural, or mixed (urban and rural) schools, depending on their geographical distribution in the district. On analysis no significant difference in the tuberculin reactor rate was to be noted in children of corresponding ages attending any one of the above three groups of schools.

Reactor Rate and Location of Scholars' Homes.—An inquiry was then made into the geographical distribution of the home of each schoolchild. The scholar's place of residence was classified into one of three types of homes—urban, rural, or mixed. Here again no significant difference

TABLE II

Age	1 Jelly-Positive	2 Mantoux-Positive	3 Tuberculin-Positive (1+2)	4 Tuberculin-Negative	5 No Mantoux Done. Jelly-Negative	6 % Positive Reactors $\left(\frac{3}{3+4+5}\right)$
5	110	15	125	466	44	19·68
6	114	20	134	438	24	22·48
7	127	28	155	390	27	27·09
8	129	37	166	312	33	32·48
9	142	44	186	266	14	39·9
10	192	38	230	268	14	44·92
11	182	29	211	245	24	43·95
12	214	35	249	259	26	46·62
13	245	56	301	217	24	55·53
14	221	41	262	165	19	58·74
15	100	7	107	46	5	67·72
16	48	7	55	11	1	
17	23	4	27	2	0	
18	4	0	4	0	0	

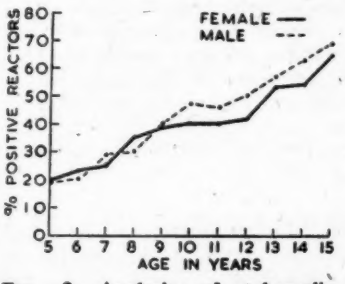

FIG. 2.—Analysis of tuberculin levels in males and females.

was observed in the reactor rate in children of corresponding ages living in a home in an urban, a rural, or a mixed area.

Value of 1/100 Mantoux Test.—An attempt was made to assess the value and limitations of the second tuberculin test (Mantoux 1/100) in determining the magnitude of any further group of reactors who failed to show a positive response to tuberculin jelly. As is shown in column 5 of Table II, a small number of children in each age group who were negative to a jelly patch test failed to attend for the subsequent intradermal injection of 1/100 old tuberculin. The total number of Mantoux reactors in column 2 of Table II was therefore adjusted by computing the proportion of expected Mantoux reactors among each age group of jelly-negative children who failed to attend for their intradermal test, and adding these to the number of previously determined Mantoux reactors (see column 2, Table II).

The proportion of children who reacted to the Mantoux test after failing to respond to tuberculin jelly varied from 3% to 11.4% (Table III).

TABLE III

Age	Adjusted Total Mantoux Reactors	Total Children	% Jelly Negative but Mantoux Positive
5	19	635	3·0
6	24	596	4·0
7	34	572	5·9
8	46	511	9·0
9	48	466	10·3
10	41	512	8·0
11	33	480	7·0
12	39	534	7·3
13	62	542	11·4
14	45	446	10·0

Radiological Findings

The results of the radiological examination are shown in Table IV. One of the most striking findings was in the group of 14 children with radiological evidence of probable bronchiectasis, 11 of whom had either a complete absence of symptoms or symptoms of so trivial a nature as to be regarded as insignificant by the parents.

TABLE IV

	No.	Per 100,000
Total children *x*-rayed	5,552	
Active primary pulmonary tuberculosis* ..	9	162
Pleural thickening	4	72
Probable bronchiectasis with/without atelectasis	14	252
Pulmonary or hilar calcification	18	324
Abnormalities of cardiac mass	2	—
Congenital bony anomalies	3	—

*Includes grossly enlarged hilar glands, epituberculosis, or lobar collapse due to glandular pressure on a bronchus.

Observations

One very surprising feature that emerged from the survey was the high tuberculin reactor rate noted in the early years of school life, and indeed throughout the entire life of the schoolchild in this area. These figures are considerably higher than those of Jones-Davies (1950) in the County of Radnor, Heimann and Paterson (1945) in Bournemouth, Fleming (1943) in Glasgow, and Thompson (1947) in Ealing. Moreover, what is perhaps even more remarkable is the difference between our findings in the Calder Valley area and those of two wholly urban areas in Yorkshire—Hull and Leeds—where similar investigations were conducted (McDowell, personal communication) (Table V and Fig. 3).

TABLE V

Age	Leeds Children	% Positive Reactors	Hull Children	% Positive Reactors	Calder Valley Children	% Positive Reactors
5	314	9·55	280	11·07	635	19·68
6	301	8·97	311	13·18	596	22·48
7	287	18·82	300	17·33	572	27·09
8	289	18·68	319	19·43	511	32·48
9	307	23·45	293	21·5	466	39·9
10	315	25·4	352	32·38	512	44·92
11	306	30·72	291	31·27	480	43·95
12	296	33·78	305	33·77	534	46·62
13	290	37·59	274	36·86	542	55·53
14	296	43·24	327	40·06	446	58·74
15	—	—	—	—	158	67·72

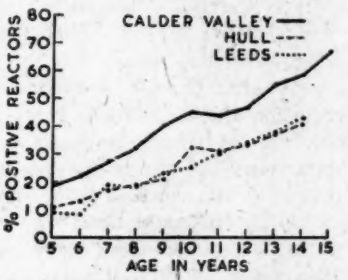

FIG. 3.—Comparison of results obtained in the Calder Valley with those of Hull and Leeds.

It will be noted that in the Calder Valley public health area the tuberculin reactor rate is between 9 and 19% higher in each age group than in Hull or Leeds. This remarkable finding is dealt with below. Generally one has been led to expect a much higher incidence of reactors in an urban area, where human pulmonary tuberculosis is a greater risk owing to overcrowding and poverty (Bradbury, 1933; D'Arcy Hart and Payling Wright, 1939).

Possibly the explanation of our findings lies in the relatively primitive methods of dairy farming which are prevalent in this area of the West Riding. Herds are often tiny, consisting of as few as 6 to 20 beasts. In many instances most of these small herds are retained by the owner-farmer for a season and then sold privately or on the market. Such cattle, because of their temporary ownership, are termed "flying herds," and anything more than a cursory supervision of the cattle is in these circumstances difficult. Moreover, the owner-farmer bulks his liquid milk into churns and often operates simultaneously as manufacturer, wholesaler, retailer, and transporter of his commodity. As much as 80% of all milk consumed in the Calder Valley area is untreated, only a very small proportion being tuberculin-tested or pasteurized, apart from that now supplied to the schools under the milk-in-schools scheme. Furthermore, we understand (Ministry of Agriculture, personal communication) that about 40% of cows in this area are positive tuberculin reactors. In the two urban regions of Leeds and Hull, however, both medical officers of health inform us that virtually all milk consumed in these towns is either tuberculin-tested or subjected to heat treatment.

In the small population of the Calder Valley area it is impossible to estimate accurately the incidence of bovine infection, and we submit the statistics in Table VI without comment.

TABLE VI

	Notified Cases of Primary Abdominal Tuberculosis	Cases per 100,000 of Population
Calder Valley	4	7·02
Leeds	7	1·39
Hull	7	2

An additional point meriting brief consideration is the notified incidence of respiratory tuberculosis (245 per 100,000) in the Calder Valley area as compared with the County Borough of Leeds, where the corresponding morbidity rate is much higher, being 507 per 100,000. Yet despite the difference in the pulmonary tuberculosis notification rates in these two areas we have evidence that the former region has a substantially higher tuberculin reactor rate in each age group.

Furthermore, we have observed a much lower radiological incidence of pulmonary calcification in the Calder Valley (0.324%) than in the Leeds survey (1.05%). We must, therefore, endeavour to reconcile the apparent paradox of a low radiological pulmonary calcification rate and high tuberculin reactor rate in the Calder Valley with the higher pulmonary calcification rate and lower reactor rate of Leeds.

We think that there is some circumstantial evidence to suggest that the high reactor rate in the Calder Valley is largely due to the high percentage of infected and untreated milk which is consumed by the child population in their homes, etc., and outside the milk-in-schools scheme. These conclusions are similar to those of Couts (1947), who was also impressed by the high reactor rate in children in the rural areas of Cambridgeshire as compared with that of children in the town of Cambridge, where only treated milk was consumed.

Conclusions

In the Calder Valley public health area 58.74% of children are tuberculin reactors by the age of 14. No significant difference in the reactor rate was noted between the sexes, or among children attending urban, rural, or mixed schools, or those residing in homes geographically situated in urban, rural, or mixed areas. It is suggested that this high incidence may be due to bovine infection from the consumption of untreated milk.

In the five-year to seven-year age groups the Mantoux 1/100 intradermal test succeeded in revealing only a further 3% to 6% of reactors. Among the older age groups (8–14 years) 7% to 11.4% of children negative to tuberculin jelly gave positive reactions with 1/100 old tuberculin.

Radiological evidence of active primary or pulmonary tuberculosis was noted in 0.16% of children in the Calder Valley.

We wish to thank the Medical Research Council for making this survey possible and the West Riding County Council and the Leeds Regional Hospital Board for their financial assistance and encouragement. Our thanks are also due to the local educational staffs, the health visitors, and the clerical personnel for their assistance.

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Medical Memoranda

A Case of Prostatic Abscess

The rupture of a prostatic abscess into the rectum has been regarded as a dread complication which should be prevented by surgical intervention. This view is often expressed in surgical textbooks (Wakeley and Hunter, 1943; Attwater, 1948; Bailey and Love, 1948; Romanis and Mitchiner, 1948; Riches, 1950), and it also appears in textbooks of venereology (Marshall, 1944; McLachlan, 1947; Willcox, 1950).

On the other hand, venereologists who have had experience of this admittedly rare contingency have noted (Burke, 1940) that the patient whose prostatic abscess points and is allowed to open into the rectum does extremely well and usually makes an uninterrupted recovery: proctitis, either gonococcal or non-specific, may be a sequel, but with penicillin and the sulphonamides this is now prevented or rapidly cured.

Because of these divergent views the following case report may be of interest.

CASE REPORT

The patient, a bachelor aged 44, attended the special treatment centre at St. Bartholomew's Hospital because of a urethral discharge. He had been treating this at home for seven months by irrigations with potassium permanganate solution.

In the last few weeks he had experienced gradually increasing frequency of micturition, burning on micturition, and pain on defaecation. Just before his attendance he was passing urine eight to ten times each night.

Examination showed a thin, ill, and apprehensive patient. His temperature was 99.4° F. (37.4° C.), pulse 100. He had a purulent urethral discharge, smears from which showed many leucocytes and mixed organisms. No gonococci were grown on urethral culture. Examination of a wet film of the urethral discharge in saline showed no trichomonads. Blood Wassermann and Kahn reactions as well as the complement-fixation test for gonorrhoea were negative. Rectal examination showed a large, boggy, tender mass protruding into the rectum. On micturition the urinary stream was narrowed; both glasses of urine were hazy, with much debris in the first. A diagnosis of prostatic abscess was made.

As the patient refused admission to hospital he was started on out-patient treatment with procaine penicillin, 600,000 units being given daily by intramuscular injection: in addition 5 g. of "sulphatriad" was given daily by mouth. He was instructed to rest at home in bed. By the second day his temperature was normal and remained so. By the third day he felt easier, the frequency had decreased, and defaecation was painless. Rectal examination showed the prostate to be less tender but larger than before. His urinary stream was still narrowed, and both glasses of urine contained pus, the second being completely opaque.

The patient was persuaded to enter hospital. The procaine penicillin was continued in the same daily dosage, and supplementary crystalline penicillin, 250,000 units daily, was administered by intramuscular injection. Sulphatriad was continued, 1 g. being given four-hourly.

On the patient's sixth day in hospital rectal examination showed the abscess to be larger, soft in the centre, and pointing into the rectum. The stream on micturition had narrowed further, but the urine was now clear.

On the seventh day surgical drainage was considered in order to prevent rupture of the abscess into the rectum; but the same day the patient passed "about a cupful" of pus when going to stool, showing that rupture had occurred.

Rectal examination the following day showed the prostate to be smaller and firmer, particularly the left lobe; pus was

seen on the examining finger. Proctoscopy showed reddened mucosa overlying the prostate; no orifice was seen, but some pus was present in the rectum.

By the tenth day after admission there was no pus in the rectum or any evidence of proctitis. On the fourteenth day the patient was discharged. He had then had, in the ward, a total of 8,400,000 units of procaine penicillin, 3,500,000 units of aqueous penicillin, and 84 g. of sulphatriad.

The patient continued to improve after discharge from hospital. After 12 weeks, rectal examination showed no abnormality apart from slight prominence and firmness of the right prostatic lobe. Prostatic fluid was normal, and the patient had gained 28 lb. (12.7 kg.) in weight. In early February, 1950, 16 weeks after rupture of the abscess, the prostate was normal on rectal examination.

COMMENT

At the time of rupture the urine was clear, suggesting that the abscess was not draining per urethram, and that, owing to this natural localization, subsequent fistula would be unlikely. It is at this stage that surgical drainage into the rectum may even be the treatment of choice.

In this case the large dosage of penicillin and sulphonamides would appear to have controlled the initial infection and prevented a proctitis subsequent to rupture.

We wish to express our thanks to Mr. A. J. King, senior physician to the Whitechapel Clinic of the London Hospital, for his helpful advice.

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Testing for Serratus Anterior Weakness

In testing for paralysis of the serratus anterior the patient is often asked to raise the arms forward or to push against a wall. These tests will demonstrate weakness if total paralysis of the serratus anterior is present, as the scapula will wing on any effort involving abduction or flexion of the arms. I have observed, however, that, in many cases in which only partial paralysis is present, if these tests are applied the winging of the scapula may unconsciously be so successfully disguised by the patient as to be almost non-existent.

If this ability on the part of the patient to mask weakness is not recognized the muscle may be passed as normal, when, in fact, partial paralysis is present. I found that by instructing the patient to adduct the scapulae before performing the test this ability to disguise winging was lost, and any weakness present immediately became apparent. This is because adduction of the scapulae will relax, by reciprocal inhibition, the abductors of the scapulae, of which the serratus is one, and, being relaxed, the serratus becomes weaker.

Figs. 1–4, photographs of a case of partial paralysis of the serratus anterior, illustrate this ability to disguise weakness and how it may be shown up by adduction of the scapulae before performing the test.

In Fig. 1 the patient, without any previous instruction, has been asked to raise his arms forward. In Fig. 2 he is

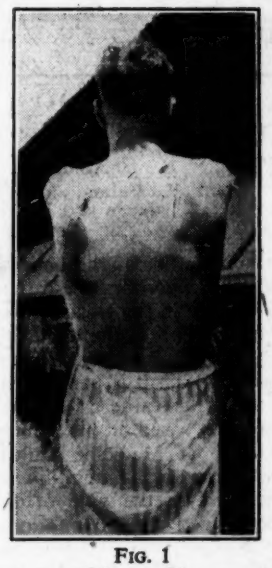

FIG. 1

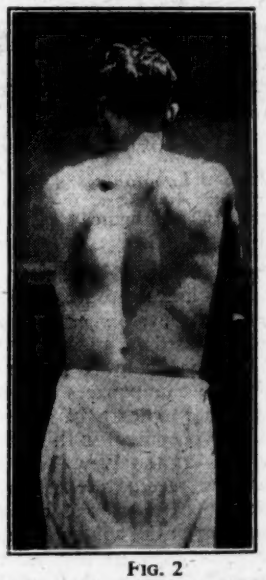

FIG. 2

FIG. 1.—Patient with arms raised forward. FIG. 2.—Patient pushing against a wall. Very little winging is apparent.

FIG. 3

FIG. 4

FIG. 3.—Patient pushing against a wall, having first adducted scapulae. Gross winging. The whole scapula has come away from the chest wall, and fingers may easily be inserted under the vertebral border. (There is also paralysis of lower trapezius.) FIG. 4.—Same patient as in Figs. 1, 2, and 3, showing amount of abduction possible.

pushing against a wall. He performs these tests with a slightly rounded back and with the scapulae wide apart. In neither case are there signs of much weakness in the serratus anterior. However, when the patient was instructed to adduct the scapulae before pushing against the wall, the result was as shown in Fig. 3. Weakness immediately becomes apparent.

In the best test for serratus weakness—flexion of the arms under resistance—weakness cannot be disguised.

Fig. 4 shows the amount of abduction a patient may accomplish by trick movements in spite of the presence of extensive paresis. This abduction can be carried out with scarcely any interruption in the rhythm of the movement.

I am indebted to Mr. C. H. Hembrow, of Melbourne, for the explanation of the observation that adduction prior to movement will show up weakness.

CYNTHIA M. WALKER,
Australasian Physiotherapy Association.

Reviews

SURGERY OF THE BRAIN

Frontal Lobotomy and Affective Behavior: A Neurophysiological Analysis. By Professor John F. Fulton, M.D. (Pp. 160; 20 figures. £1 1s.) London: Chapman and Hall. 1952.

There can hardly be a clearer demonstration of the dependence of clinical progress on laboratory experimentation than the recent developments of neurosurgery in the treatment of mental disorders, and, since the introduction of the operation of frontal lobotomy was initially based on certain experiments on chimpanzees by Professor Fulton, it is particularly interesting to learn his assessments of its value as a therapeutic procedure. In this small volume, based on his Salmon Lectures at the New York Academy of Medicine, Fulton discusses recent attempts (by ablation or stimulation of different parts of the cortex) to analyse the role of the cerebrum in the control of autonomic activity and emotional behaviour, and he also offers a brief survey of the results obtained in human patients by various operative techniques such as leucotomy and topectomy.

Much of the information presented in these lectures seems to be based on unpublished work, and for this reason it is somewhat difficult to assess its value. Particularly is this so in the case of unpublished work by candidates for the Ph.D. degree. Indeed, this may tempt the reader to some mild expostulations, for the experimental study of affective behaviour in animals is a peculiarly difficult subject and hardly appropriate for young undergraduates who are only just beginning to learn the arduous discipline of research. Here, perhaps, is one explanation of the discordant results which have been reported by different investigators. Another explanation, as Fulton points out, is to be found in the lack of proper anatomical control of the experimental material.

A third explanation (which certainly needs to be more thoroughly emphasized) depends on the failure by some physiologists to recognize how variable may be the relative extent of cortical areas in different animals—even of the same species. To these uncertainties in experimental neurology must be added the uncertainties inherent in operations on the human brain. For, even under the best conditions, it is well-nigh impossible for the neurosurgeon to determine without a post-mortem study the precise extent of the lesion which he has made. Thus there is good reason for maintaining a cautious attitude towards recent developments of (so-called) psychosurgery, and Fulton's survey makes this very evident.

W. E. LE GROS CLARK.

PHOTOGRAPHY OF PATIENTS

The Photography of Patients. By H. Lou Gibson, F.B.P.A., A.P.S.A. (Pp. 118; illustrated. £2). Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1952.

The author of this monograph has been wise in restricting his remarks to a single aspect of medical photography—namely, the recording of patients. In this way he has been able to compile a practical handbook free from irrelevant theory. The scope of medical photography has enlarged so considerably that many are now alarmed at the amount of knowledge and equipment which seem necessary before a start can be made.

Despite the establishment of centralized photographic departments in hospitals, there will remain many doctors with a keen interest in photography and with a desire to apply this to their work. Here then is a book for them: the essence of good medical photography, shorn of unnecessary complications, is presented in a clear and readable manner. The author has been careful to visualize the restricted equipment available to many of his readers, and without belittling the subject he has set standards which can be reached and maintained with simple cameras and lighting.

Although most of the examples are already familiar to photographers, the main points are convincingly illustrated and colour is used with admirable restraint. There are other monographs in the series, but this one alone will encourage the "doctor-photographer" and will help others to understand illustrative procedures. Finally, those who are called upon to teach medical photographic techniques are offered a foundation on which to base their practical demonstrations.

PETER HANSELL.

SOCIAL MEDICINE

The Advance to Social Medicine. By René Sand. (Pp. 655. £2 2s.) London: Staples Press. 1952.

Had Lord Nuffield been in a position at the outset of his career to endow a university chair of motoring, and had he chosen to be guided by the researches of the professor in developing the motor industry, by now no doubt we should have had a considerable body of published work, culminating in a large volume under some such title as "The Advance to Mechanized Travel." It is not certain that we should also have had a motor-car. Yet it is not invariably the fault of our professors if their subjects occasionally get out of hand, still less if they start so. Since its recognition by the universities, social medicine has been handicapped by its ambiguous origin, which left room for doubt whether it was to be regarded as a new and significant development in medical science, or as a contemporary expression of the humanitarian tradition which might equally well have been located in the faculties of arts or social science.

The publication of a translation of Professor René Sand's well-known book is unlikely to resolve such doubts in the minds of most English readers. It consists in a series of historical essays on the medical profession, hospitals, personal hygiene, public health, social hygiene, industrial medicine, and social assistance. In the final section, "The Advent of Social Medicine," Professor Sand has taken great pains to record carefully, if uncritically, attempts made by various authors to define and clarify the conception of social medicine. If the reader feels that the result is not entirely successful, it is no criticism of the quality of the essays, which reflect the scholarship and wide sympathy to be expected from their author. But it will help to avoid confusion if the statement that "social medicine is the final flowering of the preventive and curative art" is recognized as a comment on the humanitarian tradition in medicine. It need hardly be said that it would be premature, to say the least, if offered as a judgment on the achievements of a number of young university departments.

THOMAS MCKEOWN.

ASTHMA

The Management of Bronchial Asthma: A Guide to Treatment. By Herbert G. J. Herxheimer, M.D., L.R.C.P.Ed., L.R.C.S.Ed., L.R.F.P.S. (Pp. 107; 16 figures. £1 2s. 6d.) London: Butterworth and Co. 1952.

This is a personal and practical book describing in detail the methods of treatment and investigation used and found most effective by the author. If a diagnosis of asthma cannot be made with certainty by other methods, Dr. Herxheimer recommends the use of a histamine-inhalation test. If the patient is able to breathe a 3% aerosol of histamine phosphate from a closed circuit for three minutes, asthma cannot be present. If the patient is asthmatic a severe attack develops in 12–60 seconds. This is a severe if not heroic test, and is not likely to be used except by persons trained in spirometry. In the same way the author has attempted with some success to carry out hyposensitization by repeated inhalations of

increasing doses of allergens from a closed circuit, but he does not minimize the dangers of this method. He states that he has used it because with the normal method of hyposensitization by injections he cannot tell if he is getting an effect. This seems an unusual statement, for it is a regular finding, at least in Britain, that as hyposensitization progresses so do the skin-test reactions to the allergens used gradually diminish in size. Again, in the opinion of the reviewer the simpler hyposensitization by subcutaneous injections in truly allergic cases gives excellent results, and it must be rare that Dr. Herxheimer's more heroic methods are necessary.

A most important section of the book is that on the use of drugs in asthma. The author emphasizes that the dosage of any drug depends on the individual and on the severity of the attack. He states that it is better to give a large initial dose than repeated small ones, and any drug should be pushed in dosage until a beneficial effect is obtained or toxic signs appear. With this the reviewer is in full agreement, although of course on the first occasion on which the patient takes any particular drug it should be used cautiously.

To sum up, this little book covers its title subject very well indeed, and it has the imprint of experience on it as well as of thought and common sense. Anyone who reads it cannot fail to be helped in his understanding and management of this condition.

C. J. C. BRITTON.

BLOOD GROUPS AND THE LAW

The Human Blood Groups Utilized in Disputed Paternity Cases and Criminal Proceedings. By P. H. Andresen. (Pp. 115. £1 5s. 6d.) Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1952.

The author, who was formerly chief of the serological department of the University Institute of Legal Medicine at Copenhagen, Denmark, has had wide experience of the practical application of blood-group serology to medico-legal problems, and is well qualified to write on forensic serology. The book is not intended for the specialist in serology, but for the practising physician or lawyer who requires an introduction to the subject. It should fill this role admirably, and it should also appeal to a large body of student readers who require a short account of the subject without being submerged in masses of references to papers of no immediate interest to them. Many of the excellent standard works are too large and too detailed for the readers to whom this book is addressed, and it supplies the answer to a definite need. Such a book would be useless unless clearly written, and this book certainly is both clear and concise. The text is largely theoretical and suffers from the fact that there is no section on technique. It could be substantially improved by such an addition, unless the author feels that it is better that his readers should not attempt the practical side of the subject.

The chapter on the application of blood grouping according to Danish Law is interesting to the reader in Britain, where the courts have no power to compel the parties to affiliation proceedings to submit to blood tests but where such a step was contemplated. It is interesting to note that in Denmark a mother may be convicted of perjury if she maintains in affiliation proceedings that only one man could possibly be the father of her child and this is disproved serologically.

The author gives some details of medico-legal organization in Denmark, and the system whereby all medico-legal laboratory work is concentrated in an institute completely independent of the courts and of the police has much to commend it. He refers to the use of blood grouping in the examination of blood, saliva, and seminal stains, and emphasizes the caution which should be exercised when interpreting the results. While the ABO and MN systems are adequately discussed, and reference is made to some of the more recently discovered factors, it is felt that insufficient space has been devoted to the Rh blood groups, which are of such immense medico-legal and clinical significance, and about which sufficient is now known to use them in cases of disputed paternity. On the whole this is a useful little book.

GILBERT FORBES.

MALARIA

Malaria: Basic Principles Briefly Stated. By Paul F. Russell, M.D., M.P.H. (Pp. 210; 64 figures. £1 15s.) Oxford: Blackwell Scientific Publications. 1952.

The many important advances made in malariology in recent years, notably in regard to the life cycle of the causal organism, the chemotherapy of the disease, and its control by residual insecticides, have emphasized the need for a concise and up-to-date authoritative survey of all the data available. To summarize the principles of malariology in a few score pages is indeed a formidable task, but there is no one more fitted to undertake it than Dr. Paul Russell, whose professional life has been devoted almost entirely to the study of malaria and its many problems in such diverse countries as the southern United States, the Philippine Islands, Malaya, India, and Italy. During this period he has taken part in a number of outstanding laboratory and field researches, more particularly in the epidemiology of the disease and the development and application of control measures in rural areas.

The book is made up of five sections, devoted respectively to the parasites of malaria; its pathology, clinical course, and therapy; the mosquito vector; epidemiological problems; and measures of prevention and control. The amount of information packed into so small a compass is truly amazing, and the author has reduced the tabulation of essential facts to a fine art. One of the most remarkable instances of this is the inclusion in a single table of detailed information about the sphere of influence, time and place of biting, preferential feeding habits, indoor and outdoor resting places, and larval habitat of the fifty major anopheline vectors of the world.

Dr. Russell's monograph admirably fulfils the purpose for which it has been written. It will be found especially useful by practitioners and health officers in malarious countries who are without facilities for consulting the many articles on the subject which appear from time to time in scientific journals throughout the world. The book is well produced and is profusely illustrated with plates, charts, and a number of photographs selected from the author's own extensive collection.

G. COVELL.

DUBLIN—A CITY WITH A TRADITION

Dublin 1660–1860. A Social and Architectural History. By Maurice Craig. (Pp. 362; 80 illustrations. £2 2s.) London: The Cresset Press. 1952.

Few cities of Dublin's size possess such perfect examples of the architecture of the eighteenth century. In dignity and balance of design they are unsurpassed, and one must go back to the great days of Rome to find their equal. Trinity College, the Four Courts, the Bank of Ireland (once the Parliament House) are buildings of which any city might well be proud. And each is the centre of a tradition as fascinating as the stones in which it is embodied.

Maurice Craig has gathered into one volume an account of Dublin's architecture finely illustrated and well described and, interwoven with this, the tradition which gives it life and meaning. It is the work of a lifetime and of a lover, and only those who themselves know something of the charm of Dublin can appreciate its full meaning. It is a book to ponder over rather than to read, for the wealth of material it contains can only slowly be absorbed. The author has associated its periods with the great names of Ormonde, of Swift, and of Grattan; and he is right, for it was around these great personalities that Dublin grew, and it is to them that she owes many of her greatest memories.

HENRY SOUTTAR.

BRITISH MEDICAL JOURNAL
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SATURDAY JUNE 28 1952

RENAL FAILURE

The kidneys are silent, inaccessible, and immobile; for obvious reasons they lack the glamour of some other organs, and this may explain the obscurity which enshrouds renal disorders—a darkness accentuated by semantic squabbles. Professor Platt's recent Lumleian lectures, published last week on page 1313, and this week on page 1372, avoid this pitfall and throw a welcome shaft of light into the darkness. They are on chronic renal failure and suggest a radically different interpretation of the well-known findings. Usually the kidneys as a whole are regarded as becoming progressively less efficient, the implication being that all the nephrons are involved to a greater or less extent in a generalized functional deterioration. Professor Platt argues convincingly from structural and functional evidence that this is unlikely, and he suggests that the overall loss of function is due to complete destruction of some nephrons, while the others which remain are functionally more efficient than normal.

He compares the histological appearances of the intact nephrons and the quality of the urine passed in cases of renal failure[1] and in animals from which 80% of the renal tissue has been removed.[2] In both there is glomerular and tubular hypertrophy, associated with the production of large volumes of urine of a fixed specific gravity around 1010. In both the total glomerular filtration rate is much reduced, but it can be shown that in the experimental animals there is a 40% increase in the filtration rate per weight of renal tissue remaining. This increase is presumably due to an increase of filtration rate per nephron remaining, and it is suggested that a similar increase per nephron is very probable in chronic renal failure. In a normal dehydrated man when there is an increased excretion of an osmotically active substance (such as urea, glucose, mannitol, etc.) there is a simultaneous increase in urine flow and fall in specific gravity.[3] The greater the load to be excreted the nearer the specific gravity approaches 1010, the approximate specific gravity of plasma filtrate. Under these conditions the concentrating capacity of the normal kidney has a limit: it can either excrete a small volume of concentrated urine or a large volume of dilute urine, in the latter case with a greater total excretion per minute.

Polyuria and an apparent diminished concentrating capacity are thus part of a normal mechanism for the excretion of an excess of osmotically active substances. In renal failure there must be an increased quantity of such substances to be excreted per nephron. Urea production and 24-hour excretion are within normal limits, but histologically it can be seen that there are fewer nephrons through which this excretion can take place. Professor Platt proposes, therefore, that the polyuria and fixed specific gravity of renal failure are evidence of normally functioning nephrons, each one of which is excreting an excess of urea and other osmotic substances. When the blood urea is raised (because of the fall in overall glomerular filtration rate) it is theoretically possible that the increased excretion of urea per nephron is secondary to its high level in the glomerular filtrate, and it is not possible to deduce whether there is also an increase in glomerular filtration rate per nephron. Polyuria and inability to concentrate, with little or no rise in blood urea, however, may be due to an increased excretion rate per nephron, secondary to an increase in filtration rate per nephron—a similar increase to that found in the animals, mentioned above, which had had most of their kidney substance removed.

The fact that sodium and potassium plasma levels are rarely disturbed until the terminal stages of renal failure is taken as further evidence of the remarkable efficiency of the remaining nephrons. Glomerular filtration rates of only a few ml. a minute are compatible with a normal total excretion of these electrolytes. According to present concepts of renal function reabsorption must be diminished; in the case of potassium, actual tubular excretion can occasionally be demonstrated when the amount excreted exceeds that which has been filtered. It has been shown by Professor Platt himself that the diminished reabsorption of sodium is an active and deliberate diminution and not due to tubular failure.[4] He found that in renal failure a low percentage sodium reabsorption could be increased to normal levels by a sodium-free diet.

The small diuresis which follows the ingestion of water is not incompatible with the thesis that in renal failure the remaining nephrons are hyperactive. Glomerular filtration rate remains unchanged during

1 Oliver, J., *J. Urol. (Baltimore)*, 1950, **63**, 373.
2 Platt, R., Roscoe, M. H., and Smith, F. W., *Clin. Sci.*, 1952, in press.
3 Rapoport, S., Brodsky, W. A., West, C. D., and Mackler, B., *Amer. J. Physiol.*, 1949, **158**, 433.
4 Platt, R., *Clin. Sci.*, 1950, **9**, 367.
5 Winton, F. R., *Physiol. Rev.*, 1937, **17**, 408.
6 Smith, H. W., *The Kidney Structure and Function in Health and Disease*, 1951. New York, p. 854.
7 Bradley, S. E., and Bradley, G. P., *Blood*, 1947, **2**, 192.

a water diuresis; therefore the increased excretion is due either to a diminished reabsorption of water (the current theory) or to an increased tubular secretion. In either case even a slight diuresis in renal failure indicates an increased efficiency of the remaining nephrons. If there is diminished reabsorption of water, it can be calculated that there must be an associated tubular reabsorption of a highly concentrated fluid, necessitating much more work per nephron than has ever been shown in normal conditions. Professor Platt suggests that an increased tubular secretion of water would be a far more economical procedure, and that the small diuresis found in renal failure would then be due to a reduced number of nephrons excreting a normal quantity of water per nephron. There is no direct evidence for or against this latter theory; it is, of course, another swing of the pendulum in the everlasting argument between secretion and filtration theories of the formation of urine. That filtration takes place is now based on sound evidence, but, though it is currently accepted that filtration is due to the effect of the blood pressure forcing fluid and electrolytes out of the glomerular capillaries, there are serious theoretical objections to believing that this pressure is sufficient to push the filtrate down the tubule.[5] Theoretically, therefore, tubular secretion of some fluid is not only economically desirable but hydrostatically attractive.

Professor Platt then gives some therapeutic conclusions from the evidence now available that the kidney will do the least work when excreting a fluid similar in composition to plasma filtrate. As renal work is done in concentrating the urea and diluting the salt in the glomerular filtrate, it follows that this work will be less if the amount of urea to be excreted is reduced by a low protein intake, the necessity to concentrate urea kept to a minimum by an adequate intake of water, and the dilution of salt made less pressing by a good supply of salt in the diet. The need for a low protein diet is supported by a most interesting observation, which refutes the generally accepted view that urea retention is harmless and does not cause any of the toxic manifestations of renal failure.[6] Professor Platt himself took large quantities of urea by mouth each day for 16 days, thereby keeping his blood urea at levels varying between 50 and 90 mg.%. He says emphatically that this state of affairs was "only with difficulty compatible with a day's work, the high blood urea producing a dry mouth, lethargy, and a tendency to headache."

The lectures contain one new hypothesis which it is difficult to accept. It is suggested that the anaemia of renal failure is a compensating mechanism which allows a greater quantity of plasma to be passed through the kidney per unit of blood. This idea is based on the work of Bradley and Bradley,[7] who showed that, in cases of anaemia, as the haematocrit values fell the renal blood flow decreased but the renal *plasma* flow remained unchanged. According to these findings it is clear that, when the haematocrit is lowered, there can be an increase in plasma flow only if there is, in addition, another adaptive mechanism which will prevent the usual fall in renal blood flow which accompanies such a change in haematocrit. This is stretching the long arm of teleological argument to breaking-point and will need more solid evidence before it can be considered seriously.

TUBERCULOUS INFECTION

The Tuberculin Survey Committee of the Medical Research Council is to be congratulated on the scope and timeliness of its recently published survey[1] of tuberculin sensitization in England and Wales. Elsewhere in this issue (p. 1390) we publish a report by a team of workers in the West Riding of Yorkshire who took part in the survey. It is no credit to this country that the attack on tuberculosis should have depended so much on original work done elsewhere, mainly in the U.S.A. and the Continent of Europe. The service provided by most local authorities up to 1948 was designed to fulfil the letter of the law without allowing time, money, or staff for original research, whether clinical or epidemiological. Apart from official figures of deaths and notifications, knowledge of the epidemiology of tuberculosis in Great Britain is fragmentary. A recent paper by Aspin[2] on the spread of tuberculosis in "contact" families shows how much valuable information is hidden in the old records of chest clinics. The analysis of such data, however, may present difficult statistical problems.

The Tuberculin Survey Committee points out that previous tuberculin surveys in this country have been limited either by the age group or by the nature of the community surveyed. No previous survey on a national scale gives a picture of tuberculous infection throughout the country and at the same point in time. The wider use of anti-tuberculosis vaccine may make it impossible in the future to measure by the tuberculin test the prevalence of natural tuberculous infection in the community. The object of the survey was to determine by means of the tuberculin test how many persons at each year of age from 5 to 20 had previously had a tuberculous infection. The

[1] *Lancet*, 1952, 1, 775.
[2] Ibid., 1952, 1, 502.
[3] Daniels, M., Ridehalgh, F., and Springett, V. H., *Tuberculosis in Young Adults*, 1948, London.

slight potential limitations arising from Mantoux-reversion and the pre-allergic phase have wisely been ignored. The survey was carried out in six urban areas in the southern half of England, six urban areas in the northern half, one Welsh borough, two mining districts (Durham and Maesteg), and six rural areas in England and one in Wales. The modified tuberculin jelly test was first used, and negative reactors were given an intracutaneous test with 100 tuberculin units (0.1 ml. of 1/100 Old Tuberculin). The consent rate varied from 63% to 97% and was over 80% in 10 of the 19 areas for which the figures were available. Tests were completed on 49,795 males and 44,426 females. Those examined (over 7,000 at each year of age) appeared to form a representative social sample of the population at ages 5 to 14 in each area. At ages 15 to 20 the samples were less satisfactory, since there was a preponderance of school and university students. The figures were, however, weighted by the inclusion of 3,450 men aged 18 to 20 examined on recruitment for National Service. Where these Servicemen gave a home address in a survey area the results were included in the appropriate area. Those organizing the survey took great pains to establish uniformity of technique. The distinction between positive and negative reactors is clear and consistent, and is not open to serious criticism. Because the tests were carried out by more than 30 different doctors no attempt has been made to estimate degrees of tuberculin sensitivity. The findings in different areas are reported in detail, and the areas are also combined into large geographical groups—a method which is probably valid for the northern and southern urban areas but which, as will be shown, raises some doubts when the rural group is considered.

The curves representing the percentage of positive reactors rose progressively from the age of 5 years up to that of 20. The level was consistently higher for the northern than for the southern urban groups. There was a notable difference between rural and urban areas, and this difference persisted even when Cheshire and Montgomery (the two extremes of "scatter" in the rural figures) were excluded. At the age of entry to school one in every five rural children was already infected; the southern urban areas averaged one in ten, the northern urban areas one in eight. This higher proportion of positive reactors in country children persisted throughout school life. Annual rates of infection showed little change during school life and were round about 5% in the northern urban and rural areas, and slightly lower in the southern urban areas. By the age of 14 half the children in the rural areas had been infected, nearly half of those in northern urban areas, and just over one in three in the southern urban areas. Once the school-leaving age was passed the infection rate was more than doubled in all the urban groups. In the rural areas there was a smaller rise in the rate for males and virtually no change in the rate for females. During the school years the infection rates were about the same for both sexes, but subsequently the increase seemed to be greater in males. Comparison with the Prophit survey[3] suggests that the prevalence of infection has fallen noticeably during the past 15 years.

These are significant and provocative findings. Primary infection in adolescence is known to be associated with a high morbidity. Delayed primary infection is clearly becoming more common. The present findings suggest that the sex difference in reaction to infection during adolescence is even more unfavourable to young women than is revealed in morbidity and mortality statistics. If the protective value of anti-tuberculosis vaccination is accepted, the case for its application to all school-leavers seems to be clear. The authors of the report relate the fall in the general prevalence of infection and the relatively higher rates during school age in rural areas to the greater availability of heat-treated milk in the large towns. This conclusion is reasonable and probably true. Nevertheless, the validity of the grouped figures for rural areas may be questioned. The individual figures for the urban areas comprising the groups are quite consistent, but in the rural areas there are some notable discrepancies. At age 5 the percentages of positive reactors range from 8.4 in Montgomery to 30 in Cheshire; at age 14 from 24.6 in Montgomery to 67.6 in Cheshire; at age 19 to 20 from 50 in Montgomery to 82.4 in Cheshire. At every age the lowest figures are recorded for Montgomery, whereas Cheshire shows more positive reactors than any other area, urban or rural. It is not clear why Cheshire should be exceptional. If 3,510 children and adolescents from Nantwich and Northwich constitute a fair sample of Cheshire, why has this county, with a density of 847 persons per square mile, nearly twice as many children infected with tuberculosis as Southwark, where there are 54,802 persons per square mile? Cheshire, it is true, has more cows than Southwark; but are more of those cows tuberculous, and is less of their milk pasteurized than in Montgomery? It is to be hoped that the authors of the present report will attempt to correlate their findings, not only with the figures of tuberculous infection and disease in cattle but also with the figures for human tuberculosis morbidity in the areas they have so well studied.

EPIDEMIOLOGY OF VIRUS HEPATITIS

Infective hepatitis belongs to that small but important group of infectious diseases which from time to time burst with almost explosive violence into epidemics of pandemic proportions. Unlike other diseases showing this epidemiological peculiarity, such as influenza, great increase in its incidence is fortunately not associated with any obvious increase in virulence, and the mortality rate, in the economically superior communities at least, rarely exceeds 0.2%. It may, however, apart from more persistent sequelae, incapacitate patients for weeks or months, and in the second world war it seriously affected the strategy of both the contending armies, especially in the Mediterranean theatre. Moreover, among peoples already weakened by poverty, malnutrition, and disease, epidemic hepatitis may levy a much higher toll. Recent experience teaches quite clearly that war, especially world war, creates the ideal situation for widespread dissemination of this viral disease. This is clearly shown in Sir John McNee's Harveian Lecture, published in the opening pages of this issue. As he rightly points out, the many possible causes of epidemics of jaundice—including Weil's disease, malaria, dysentery, and the enteric groups of fevers—could not be clearly differentiated before the development of the science of bacteriology. It is therefore difficult to assign to its precise aetiological agent any epidemic prior to the bacteriological era. But because of the low mortality of viral hepatitis it is possible to distinguish fairly readily epidemics of viral jaundice from those due to other agents. In the epidemic among the troops in the American Civil War 161 deaths were reported amongst 22,509 cases—that is, a fatality rate of 0.7%—and in the South African War a similarly low fatality rate was recorded amongst 5,648 cases. In the first world war also the outbreaks in Mesopotamia claimed only 46 lives out of over 6,000 cases, and in the war of 1939–45 the fatality rate among the Allied troops was about 1 in 500.

Serious epidemics of viral jaundice are rare apart from war, but isolated cases and small outbreaks were common throughout Europe between the two wars. Such outbreaks provided opportunity for important studies in epidemiology[1] and appeared to show that the disease was spread by infected droplets among close contacts. But studies on the spread of infection among the troops, and the experiments on transmission to human volunteers, show that the infecting agent is much more readily recoverable from the faeces than from nasal washings, and that transmission by faecal contamination is therefore the most probable means of epidemic spread. The association of epidemics with the presence of large numbers of flies accords well with this view. Water- and milk-borne epidemics, however, have been only rarely reported.

Sir John McNee's lecture provides a most useful summary of the growth of our knowledge of hepatitis and a salutary warning about the dangers of pandemic spread in the event of another world war. A more detailed account of the present position is also available, for the Medical Research Council has recently published a report[2] based on studies of the epidemic of infective hepatitis in East Anglia during the period 1943–7. The clinical observations were on 2,000 cases, and also included are the results of biochemical, haematological, and serological investigations, and an excellent summary of the attempts at transmission of the disease to animals and men. For the rational control of epidemics it is essential to have clearly established, in addition to knowledge of the means of spread, the incubation period, the period of infectivity, the frequency of subclinical cases, and the most reliable means of early diagnosis. Useful information on all these matters is presented in the M.R.C. report. The study of the incubation period has, moreover, clearly differentiated two varieties of the disease—namely, homologous serum jaundice due to parenteral administration of icterogenic blood, or product of blood, with an incubation period of 60 to 120 days; and the naturally acquired disease with an incubation period of 20–35 days. Despite this fairly clear-cut difference McNee adheres strongly to the view that there is only one hepatitis virus, and this is supported by the close similarity of both the clinical picture and the histology of the affected liver in the two diseases. But the weight of evidence from the human transmission experiments is in favour of two distinct viruses. Quite apart from the difference in incubation periods, there is fairly convincing evidence, despite the small number of cases tested, that a person can be immune to the homologous virus and not immune to the heterologous virus. There is, moreover, a well-established difference in the routes by which artificial transmission can be achieved. Whereas in homologous serum jaundice successful transmission has been obtained only by parenteral administration of serum, infective hepatitis can also be transmitted by spraying the nasopharynx with infected serum. The virus causing infective hepatitis has also been conclusively demonstrated in the faeces, whereas no evidence of faecal virus has been obtained in patients with homologous

[1] Pickles, W. N., *Epidemiology in Country Practice*, 1939, Bristol.
[2] MacCallum, F. O., McFarlan, A. M., Miles, J. A. R., Pollock, M. R., and Wilson, C., *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 273, 1951, H.M.S.O., London.

serum jaundice. Finally, although in the majority of cases the clinical picture of the two conditions is indistinguishable, many observers have noted a tendency for the homologous serum variety to be more severe and more protracted.

The period of infectivity in both varieties of the disease appears to be of remarkably short duration, but experiments to determine this have been very few in number. In an induced case of infective hepatitis virus has been detected three days before the first symptom and as late as eight days after the onset of symptoms, but not 31 days after the onset of symptoms. A chronic carrier state with infective hepatitis virus in the blood stream does not seem to occur. The history of homologous serum jaundice also shows quite clearly that donors of icterogenic plasma exist in whom no association with cases of infective hepatitis appears at all probable. The origin of the icterogenic agent in their blood is therefore completely obscure. If it has arisen, as McNee suggests, from ordinary infective hepatitis virus by adaptation from prolonged sojourn in the human blood, the evidence for this has yet to be put forward. Even if this were so, the stability presented by the two viruses to-day is such as to justify their classification as distinct varieties despite any possible common origin.

ORGANIZATION FOR MEDICAL MYCOLOGY

Human fungal infections are common in many parts of the world; but in Britain their study has been unduly neglected, and, skin diseases apart, cases are probably missed. This is likely to become more important, as secondary infections with *Candida albicans*, for example, now sometimes follow antibiotic treatment, and there is a growing need for more investigation and the spread of information. A number of recent developments in medical mycology in Britain can be traced back to the inspiration of the late Sir Edwin Butler. He was a medical man by training, and successively imperial mycologist in India, director of the Commonwealth Mycological Institute, and secretary of the Agricultural Research Council. In 1922, as first editor of the *Review of Applied Mycology*, Butler decided to abstract papers on mycological aspects of medical mycology in that journal, and this policy was continued by subsequent directors of the institute. When the working of the Commonwealth Agricultural Bureaux was examined by the Hankey Committee in 1942 medical mycology was one of the topics discussed. An approach was made to the Medical Research Council, which convened a conference of interested parties, and as a result the Commonwealth Mycological Institute agreed to issue in collaboration with the Bureau of Hygiene and Tropical Diseases an annual *Annotated Bibliography of Medical Mycology*, and the M.R.C. appointed a committee to advise and assist the council in encouraging research in medical mycology. During the past nine years both these projects have developed.

Towards the end of the war J. T. Duncan, at the instigation of the Medical Mycology Committee, made a survey of the mycoses of man in this country (the results were summarized in this *Journal*[1]), and that survey was one factor leading to the establishment of a Mycological Reference Laboratory within the Public Health Laboratory Service. This laboratory, situated in the London School of Hygiene and Tropical Medicine, offers a diagnostic service and maintains a collection of cultures of fungi of medical importance. The committee next turned its attention to the nomenclature of fungi pathogenic for man and animals, and a list of recommended names of these pathogens was published in 1949 as M.R.C. Memorandum No. 23. This list, which is being kept under review, will, it is hoped, be accepted as a standard for the country, and a number of editors have agreed to adopt the recommended names in their journals. Another project initiated by the committee is the study being made at Aberdeen of the status of the organisms causing actinomycosis in man and animals. The appreciation of the common interests of workers in the medical and veterinary fields led to a broadening of the committee by the inclusion of representatives appointed by the Agricultural Research Council. Stocktaking of the mycoses of this country is now being completed by a survey (sponsored by the A.R.C.) of the fungi found associated with disease in farm animals.[2]

The *Annotated Bibliography* proved its usefulness, and the last two issues have recently appeared as parts 9 and 10 of the *Review of Medical and Veterinary Mycology*. The editorial board of this unique journal of abstracts has been enlarged by the addition of the director of the Bureau of Animal Health, and it is intended that two parts shall henceforth appear each year.[3] Since its inception more than 2,000 papers have been noticed, and with increasing activity in the field surveyed all those interested in any aspect of fungi responsible for disorders in man and animals will find this journal an increasingly useful tool.

INTRAVENOUS FEEDING WITH FAT EMULSIONS

Parenteral feeding by emulsions of fat has a history of more than 50 years (reviewed briefly by A. Beauvillain[1]), but it is only during the past few years that the difficulties of preparing fat emulsions suitable for intravenous administration have been overcome. To avoid the danger of embolism, the fat, most suitably coconut oil, is emulsified under high pressure (up to 3,000 lb./sq. in.) at 70–80° C., and the emulsion is stabilized with purified lecithin made from egg-yolk or soya-bean. The size of particles in well-prepared emulsions is less

[1] *British Medical Journal*, 1945, **2**, 715.
[2] Details of this survey may be obtained from Dr. G. C. Ainsworth, A.R.C. Survey, University College, Exeter, Devon.
[3] Annual subscription 10s., post free; Commonwealth Mycological Institute, Kew, Surrey.

than 2 μ, and most particles have a size under the resolving power of the ordinary light microscope, so that they are visible only by the ultramicroscope.[2] The preparations can be sterilized by autoclaving without breaking up the emulsion.

The intravenous feeding of patients with such emulsions is now a safe procedure which has decided advantages over the administration of glucose or of protein hydrolysates. Fats do not exert an osmotic pressure, and therefore emulsions containing any desired concentration of fat may be given without danger of causing thrombosis; further, on account of the high energy content of fat more calories can be derived from a given volume of fat emulsion than from the same volume of glucose or protein hydrolysate. A 15% fat emulsion in 5% glucose provides about 1,600 calories per litre, compared with 400 calories per litre of 10% glucose alone, or 800 calories per litre of a preparation of 5% glucose + 5% protein hydrolysate + 7.5% alcohol. The use of fat emulsions in conjunction with the more conventional type of intravenous alimentation thus has the advantage of providing additional calories, particularly during prolonged periods of energy deficit, which cannot be supplied only by the administration of glucose or of protein hydrolysates. Lerner, Chaikoff, Entenman, and Dauben[3] showed, with the aid of tripalmitin labelled with radioactive carbon, that one-half of an intravenously administered fat emulsion was oxidized in 24 hours. There can be no doubt, therefore, that intravenously injected fat takes part in the body's metabolism.

Van Itallie, Waddell, Geyer, and Stare reported recently[4] the intravenous feeding of 35 patients with 10–15% fat emulsions. One-half to two litres of emulsion were given once or more often. They describe in detail the case of one patient who, on account of complete intestinal obstruction resulting from generalized peritonitis, was fed for 67 days entirely intravenously until he was able again to take food by mouth. Of the total of nearly 82,000 calories given parenterally slightly more than 42,000 calories were provided by 36 injections of a fat emulsion during the last 36 days of intravenous feeding. It seems very probable, as Van Itallie and his colleagues indeed believe, that this patient could not have survived his illness without the additional calories provided by the intravenously injected fat.

The technique of administering emulsions of fat is not different from methods of intravenous administration of other solutions, and a litre of fat providing 1,600 calories can be given in about two hours. The clearance of fat from the blood is complete after about 4 to 5 hours. The only untoward effects that have been observed in a few patients are nausea and pyrexia. It seems very probable from the published accounts that it is not the fat itself but some impurity in the stabilizing agent which is pyrogenic. Van Itallie emphasizes justly that in the light of experience already gained a discussion whether the intravenous administration of fat to patients is feasible or not is beside the point. The concern of future work must be to make these fat emulsions more readily available and to eliminate all pyrogens from the preparation.

1 *Pr. méd.*, 1952, **60**, 390.
2 Geyer, R. P., *et al.*, *J. Lab. clin. Med.*, 1948, **33**, 163.
3 *Science*, 1949, **109**, 13.
4 *Arch. intern. Med.*, 1952, **89**, 353.

VITAMIN B_{12} AND GROWTH

Growth is influenced by a multitude of factors. Attention has recently been focused upon one possible stimulant to the complex process—namely, vitamin B_{12}. Wetzel *et al.*[1] and Chow[2] claimed that oral vitamin B_{12} promoted growth in small groups of children of widely differing ages, and mostly in chronically ill or malnourished subjects. Downing[3] and recently Benjamin and Pirrie[4] have not been able to confirm those results, the former in premature infants, the latter in chronically ill children. Dr. John Yudkin, elsewhere in this issue, is also discouraging about the value of the vitamin. He reports the results of feeding liver supplement to a large group of children satisfactorily homogeneous with regard to age, sex, height, and weight. The children were all healthy as far as could be determined, and of average height and weight, and there was no reason to suppose that their unsupplemented diets were in any way deficient. Yet these children gained 20% and 40% more in height and weight than a similar control group in the short space of 13 weeks.

This liver supplement certainly contained small amounts of vitamin B_{12}, and it would be tempting to say that it was this vitamin which stimulated the growth. However, Dr. Yudkin gives strong evidence that the factor responsible was not B_{12}. Rats also showed greater growth when the liver powder was added to their diet, but not when vitamin B_{12} alone was the supplement.

A further point of interest is that height and weight do not give us a detailed picture of body size, and it would be interesting to investigate in what pattern the factor produced its changes. In other words, for example, where was the weight put on—in muscle, bone, or fat? Those engaged in detailed growth studies tell us they are able to differentiate in this way, and it may be that different growth factors produce different patterns of growth.

This work raises also the question whether it is desirable to feed a growth factor to children to "make them grow": this would apply to the presumed undersized or malnourished child, or indeed to any child, to make him grow "big and strong," as has been suggested. It is important to keep before us the concept of growth as a form of change influenced by delicate interrelationships of many diverse factors. The change stops when it has attained its terminus of maturity, and this terminus is probably fixed. It is doubtful whether there is any evidence that causing temporary accelerations of the change from time to time therapeutically will benefit the individual or alter the form of his final maturity.

1 *Science*, 1949, **110**, 651.
2 *J. Nutrit.*, 1951, **43**, 323.
3 *Science*, 1950, **112**, 181.
4 *Lancet*, 1952, **1**, 264.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

CLINICAL EFFECTS OF EXPOSURE TO HEAT AND SUNLIGHT

II. HEAT EXHAUSTION AND SUNBURN

BY

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HEAT EXHAUSTION

Heat exhaustion commonly results from exposure to moist heat. The heat-controlling mechanism is not seriously affected and there is thus no great rise in body temperature. There is, however, always a change in the body water/salt balance, and severe cases are complicated by the appearance of circulatory insufficiency and shock. The degree to which the cardiovascular system is involved is variable, but such involvement may be profound, especially in hot moist working conditions.

Most cases in the Tropics occur at the hottest time of the year. All ages and both sexes are susceptible. Under suitable conditions the syndrome appears in both visitors and indigenous populations.

Predisposing factors are similar to those in heat hyperpyrexia (see Part I). Of particular importance are heavy muscular exercise, inadequate intake of water and salt, loss or absence of acclimatization to hot working conditions, and concomitant febrile illnesses, especially malaria and pneumococcal infections. Dry-bulb temperatures of the order of 100° F. (37.8° C.) or over, associated with wet-bulb temperatures of 90 to 97° F. (32.2 to 36.1° C.), are apt to precipitate the syndrome.

It may be difficult to determine from the history whether there is obvious water or salt deficiency, but consideration of the tremendous losses of fluid and salt that may occur during heavy work in hot conditions, such as are found in mines, will usually indicate the presence of deficiencies which can be confirmed by examination of the blood and urine.

Clinical Picture

The onset may be abrupt, but there is often a prodromal period of several days in which the patient complains of progressive headache and anorexia, frequently associated with nausea and mild vomiting. Fleeting cramps, particularly in the muscles of the calf and foot, are common. There is no change in sweating, but the urinary output is often low. Giddiness associated with unsteadiness of gait may be observed shortly before the onset. Visual and aural disturbances such as "spots in front of the eyes" and "ringing" in the ears are common.

On examination the patient is usually exhausted, restless, and anxious. In more serious cases he may be lightly comatose. Loss of consciousness and other central nervous system signs are, however, minimal compared with those in heat hyperpyrexia and are of short duration, responding readily to treatment. The oral temperature may be normal or slightly raised. The rectal temperature is seldom higher than 102° F. (38.9° C.). The skin is cold, moist, and pale as a result of vasoconstriction. Sweating may be profuse. The skin is inelastic from dehydration, the facies is pinched, the eyes are sunken, and the cheek bones are often very prominent. Breathing is rapid and shallow. In comatose cases it may be deep and stertorous. If vascular failure has developed there may be evidence of pulmonary oedema.

Most patients vomit freely. The vomiting is not always accompanied by nausea and may be very intractable. Large quantities of chloride may be lost in this way. Vomiting usually precedes the onset of muscular cramps, which occur most commonly in the legs, thighs, and arms. Cramps develop in abdominal muscles only in severe cases. Occasionally there may be carpopedal spasms resembling those of tetany.

The pulse rate is nearly always very fast, though occasionally there may be noticeable bradycardia. In mild cases the systolic pressure may be below 90 mm. Hg, but the pulse pressure is low, since the diastolic pressure often remains high in relation to the systolic because of the prevailing peripheral vasoconstriction. Sudden change of posture from lying to standing has a severe effect; the pulse races, the blood pressures fall, and the patient may faint. When medical shock appears the pulse rate rises, sometimes to 150 to 200 beats a minute. The diastolic blood pressure falls rapidly to a point at which it may not be measureable. The systolic pressure falls less in proportion, so that the pulse pressure tends at first to rise. In severe cases, however, the pulse may be imperceptible at the wrist. Loss of circulating blood volume is indicated by haemoconcentration, the haemoglobin and red-cell count rising by as much as 150% with an associated increase in blood viscosity.

Urine.—The urinary volume is reduced, often to about 500 ml. or less in 24 hours. When shock has appeared there may be complete suppression of urine, which may be transitory or persist, leading to acute uraemia. The urinary concentrations of urea and chloride are low; in severe cases there may be no chloride present. The concentration of fixed bases is also usually low. Chloride loss is excessive in cases in which vomiting is severe; sodium loss may be excessive when diarrhoea is predominant. For practical purposes it is enough to judge the mineral deficiency in terms of urinary chloride concentration only. The urine may contain albumin and hyaline and granular tubular casts. In severe cases the latter may be present in large numbers.

Blood.—The whole blood and plasma chloride concentrations are low in most cases, especially where vomiting has been severe. Blood urea nitrogen is frequently raised above normal and may attain a figure of about 100 mg. per 100 ml. even in cases in which there is no obvious renal failure. When acute uraemia develops the blood urea nitrogen rises rapidly as the uraemia develops.

Sweat.—The secretion of sweat is not restricted, as it is in hyperpyrexia. In most cases the chloride content is raised and the amount of salt lost is thus considerable. The high chloride concentration of the sweat persists for some time into convalescence.

Course and Prognosis

The death rate is low in uncomplicated and correctly treated cases. Shock greatly increases the risk to life, but with adequate and prompt treatment even the shocked patient will usually recover. When urinary suppression has developed the outlook is bad. Even in the most severe cases, however, recovery is usually at the most a matter of a few days, and with correct treatment may be only a matter of hours.

Provided the water/salt balance is adjusted and, where necessary, acclimatization is re-established there is no tendency to recurrence.

Diagnosis

A history of exposure to heat, particularly under hot working conditions, must be demonstrated. Predisposing factors are usually evident. It is essential in all cases to exclude malaria (especially falciparum) and other febrile illnesses. The presence of concurrent disease does not affect the treatment required for adjustment of the disturbances of water/salt balance or shock.

Treatment

Heat exhaustion is essentially a condition of dehydration associated with mixed water and salt deficiency, complicated in severe cases by vascular failure and acute reduction of effective blood volume. Treatment must aim at replacing fluid and salt as required and readjusting the blood volume.

All cases should be treated in bed. Mild cases will recover very quickly and may be allowed to return to work in a few days. They should be given a fluid diet in the first instance which ensures that they receive 5 to 6 pints (2.8 to 3.4 litres) of fluid in the first 24 hours. The salt requirement, normally 25 to 40 g. a day, can be administered orally in drinks or capsules. In severe cases parenteral replacement of fluid is of immediate importance. If the degree of dehydration is severe and there is extreme salt deficiency the first injections of isotonic saline must be given quickly—for example, the first pint (half litre) in 15 minutes, the next in 20 minutes, and the third in an hour. Thereafter fluid can be given at the rate of a pint every four to six hours up to a total of five pints (2.8 litres) in the first 24 hours. After three pints (1.7 litres) of isotonic saline have been given it is better to continue if necessary with either alternate pints of isotonic glucose and isotonic saline or a mixture of equal quantities of these solutions. An initial pint of plasma administered in half to one hour is needed in shocked patients. Conscious patients should be encouraged to drink saline fluids by mouth. A fluid intake/output account should be kept in all cases, and the urinary chloride content should be checked at intervals during infusion.

Recovery in most cases is very rapid. Parenteral administration of fluids is seldom required after the first 24 hours. In an adequately treated case the plasma chloride may return to normal values in under 48 hours. The urinary chloride concentration recovers more slowly.

SUNBURN

Sunburn is a reaction which follows exposure to ultra-violet light of wavelengths of 2,900 to 3,200 A. Heat waves play no part in ordinary sunburn, which may readily appear after exposure to "cold" light, such as that reflected from water or snow. Heating the skin may, however, make it more sensitive to the active light.

The degree of burning depends on many factors, including the duration and intensity of the exposure. The area exposed is important; certain areas, such as the skin of the back and thighs, the back of the neck, and the popliteal fossae, are particularly susceptible. Blonds are affected more rapidly and more severely than brunettes or coloured individuals, in whom the pigmentation affords protection. Some protection is also provided by sweating and by slow acclimatization, which intensifies the degree of skin pigmentation, especially in brunettes, and to some extent increases the thickness of the horny layers of the skin. There is some evidence that the physiological state of the individual may affect the reaction to exposure; sunburn, for instance, is more easily acquired in the immediate premenstrual state.

Clinical Picture

Acute sunburn follows exposure after a latent period of half an hour to some hours. The first sign is erythema of the exposed parts, which may be only slight and transient, accompanied by some itching and a burning feeling in the affected areas. More severe cases develop acute erythema with oedema of the skin, which after a few hours develops vesicles and bullae filled with clear fluid. The affected area is surrounded by a considerable erythematous flare. If the skin of the face is affected there is often concomitant severe conjunctivitis. In the absence of secondary infection, which may follow the rupture of blisters and bullae, the inflammatory reaction subsides, and desquamation and peeling follow, leaving irregular areas of hyper- and hypo-pigmentation.

Healing is usually rapid and complete, but complications are common, especially local secondary infection, which may be severe and resemble impetigo. The affected area often shows subsequent increased sensitivity to active light, which may persist for long periods. Herpes and telangiectases may appear in the healed areas.

Severe sunburn is accompanied by general signs and symptoms which may appear soon after the local signs become manifest, and which may be very serious if the exposure has been excessive. There may be malaise and headache, and nausea and vomiting often occur. In severe cases fever is the rule. In the worst cases fatal shock may develop. The general signs last a few hours to some days, depending on the severity of the burning and on the development or otherwise of sepsis.

Treatment

The affected area should be cleaned with calamine lotion or liquid paraffin, not with soap; antiseptic and analgesic ointments should be avoided. Calamine lotion should be applied freely. Badly affected small areas may be treated with the application of cold calamine or aluminium acetate (1%) compresses. Compresses should not be used on large areas. Blisters should be ruptured and the exposed areas painted with calamine lotion to which may be added 0.5% crystal violet. After the first inflammatory stage has subsided, calamine or zinc creams will usually rapidly heal blistered areas.

Where secondary infection is established the area should be bathed several times in the day with weak mercuric chloride (1/4,000) or similar solutions. Where sepsis is severe general treatment with antibiotics or sulphonamides, depending on the prevailing organism, may be required.

The local application of antihistaminic agents is useless, but these drugs, given orally, may sometimes relieve the itching and burning sensations which can be so troublesome.

General treatment consists of avoidance of further exposure and the administration of sedatives. Severely burnt individuals may require barbiturates or morphine. Severe sunburn must be treated like other burns and cradled and protected according to the area affected.

Prevention

The most sensible thing to do about sunburn is to avoid it. Ordinary glass will screen most of the active wavelengths. Some degree of acclimatization may be acquired even by fair-skinned individuals by regular controlled exposure, which ultimately results in increased skin pigmentation.

Considerable protection may be afforded by the use of chemical screens, which are designed to protect against both ultra-violet and infra-red light, or against ultra-violet only. Ordinary cosmetic face powders and creams have protective properties which may be specifically increased by the addition of certain substances that absorb the active ultra-violet wavelengths. These compounds include tannic acid (5%), quinine hydrochloride (5%), *p*-aminobenzoic acid (10%), and salol (10%) made up in a vanishing-cream base or dissolved in commercial spirit. Protective creams or solutions should be rubbed into the skin every three to four hours during exposure, and after swimming, especially in sea-water.

A combination of intelligent acclimatization and the use of creams will protect against sunburn, particularly in dark-skinned individuals. In the latter there is often little risk of sunburn on moderate exposure, and rapid pigmentation can be produced by treating the skin with materials which screen off the ultra-violet but not the infra-red wavelengths.

Vegetable oils act in this way, especially sesame oil. Mineral oils are not satisfactory. Thomas suggests the following as a "sun-tan" oil:

Methyl salicylate	10%
Sesame oil	50%
Liquid paraffin	40%

It should be noted that none of these creams, lotions, or oils will protect against the development of certain of the more chronic effects of exposure, especially those arising from sensitization. Care should be taken to avoid undue interference with sweating, which may result from too lavish and persistent use of protective creams and occasionally may precipitate hyperpyrexia due to interference with heat loss (see Part I).

The use of creams, etc., containing specific agents such as *p*-aminobenzoic acid must be watched carefully, since skin sensitivity to the chemical agent may be present, particularly if the area has been previously exposed to sunlight.

Chronic Effects of Sunlight

The more persistent effects of sunlight on the skin occur usually in individuals frequently exposed to the active light. They include atrophy of the epidermis, irregular patchy pigmentation and depigmentation, and solar keratitis (wart-like local hyperplastic areas which may go on to the development of basal-cell or squamous carcinoma). Many individuals habitually exposed to sunlight develop plaque-like, pinkish, excoriated, and scaly lesions varying from 1 to 3 cm. in diameter which appear on the exposed parts, especially the cheeks and the anterior region of the neck. Pruritus is common in these cases, and the plaques are often seen in association with more generalized polymorphic eruptions (summer prurigo) stimulated by exposure to the "sunburn" spectrum (2,900–3,200 A). These eruptions are exacerbated on exposure during the summer and remit in the winter. They develop especially on the face, appearing as an itchy papular vesicular eczema which may become chronic and does not respond easily to treatment.

Solar urticaria in the form of non-confluent urticarial wheals and erythema may develop after the mildest exposure to sunlight, especially in individuals who have been sensitized by previous exposure to acute sunburn. The active radiations are infra-red.

These chronic conditions are often very refractory to treatment and require specialist advice. Protection against exposure is most important. In the case of summer prurigo active rays may be filtered by suitable physical agents, such as lotions containing quinine, tannic acid, and *p*-aminobenzoic acid, but it must be remembered that sensitivity may exist to the chemical agent used.

Next Refresher Course Article.—"Quarantine and Isolation," by Dr. R. E. Smith.

Nova et Vetera

PERSONAL HYGIENE IN ANCIENT ROME

A Translation of Galen's Hygiene (De Sanitate Tuenda). By Robert Montraville Green, M.D. With introduction by Henry E. Sigerist, M.D. (Pp. 277. £2 2s.) Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1952.

Galen (130–200 A.D.) was the most voluminous medical writer of ancient times. The bulk of his surviving works, which are entirely in Greek, exceeds that of all the others combined. Apart from the Hippocratic Collection, almost all known of ancient anatomy, physiology, hygiene, and internal medicine comes from or through Galen. From him, too, we have, directly or indirectly, the bases of our medical vocabulary. Hardly any of this vast and varied literature is available in English. First in the field was the late Dr. A. J. Brock, of Edinburgh, who translated Galen *On the Natural Faculties*, a work on the general principles of physiology. Professor R. M. Green now comes next with Galen's *On Hygiene*. The writer of this review has in the press Galen *On Anatomical Procedures*, which is on practical physiology and anatomy, while Mrs. May, of Cornell University, is preparing Galen's great theoretical treatise *On the Uses of the Bodily Parts*. When we have also a specimen of one of Galen's works on treatment the English reader will have access to a fair selection of the works of the greatest medical writer of antiquity, some 1750 years after his death.

The work which Professor Green presents is concerned with living wisely and well in the purely animal sense. Beyond advising serenity of mind and such occupations as reading and study, Galen has here nothing to say as to the ordering of the mental and spiritual life. The book gives an impression of such excessive concentration on details of physical health as, in a modern man, would be regarded as pathologically valetudinarian. This is characteristic of his age and society, for Galen is advising the idle rich, and nothing that can lead to a sense of physical well-being is omitted. No minutiae of massage, of exercise, of diet, of the bath, or of repose are too small for consideration. This has the advantage of giving an insight into the habits of the wealthy in "the great age of peace" of the Antonine Emperors. Life in Rome then seemed more secure and settled than perhaps at any time or place in history.

Galen's instructions extend literally from cradle to grave. "The new-born infant should be powdered and wrapped in swaddling clothes, that his skin be made firmer than the parts within. For during pregnancy everything was equally soft, since nothing of a harder nature touched it, and no cold air came in contact with it, whereby the skin would become tougher and denser than the other parts of the body. But when the baby is born, his natural covering should be best prepared by us for exposure." (Much shortened.) So he goes on through childhood to puberty, when he especially advises various forms of exercise and a careful diet, all minutely described, for "as the trainer is the attendant of the gymnast, so the cook is of the physician."

Galen often includes case-histories. He devotes several consecutive chapters to the hygiene of old age, making this the earliest work on geriatrics. One of his patients, Telephorus, a studious man, lived to be 100. "He used to bathe twice a month in winter, and once a week in summer, and three times a month in the intervening seasons of spring and fall. When he did not bathe he was anointed about the third hour, with brief massage. Then he used to eat barley boiled in water mixed with the best raw honey, and this alone sufficed him at first. And he also dined at the seventh hour or a little sooner, eating vegetables first, and then fish or game. But in the evening he ate only bread moistened in diluted wine." (Shortened.)

The overweight patient who fell into Galen's hands was in for a severe discipline. "I have made any stout patient moderately thin in a short time by compelling him to do rapid running, then wiping off his perspiration with very soft or very rough muslin, and then massaging him maximally with diaphoretic inunctions, and then to the bath. After which I did not give him nourishment immediately, but bade him rest for a while, then to the second bath, and then gave him abundant food of little nourishment, so as to fill him up but distribute little of it to the body." (Shortened.)

Galen is anything but concise, and seldom uses two words when he can use twenty. Nor is it always possible to treat seriously his tirades against those who differ from him on some minor point, though in this work he is no more than pleasantly discursive and is less often stirred by controversy than is his wont. The translation is faithful, erring sometimes a little on the side of too exact literalness—a good fault—but it would bear far more annotation than it has received. Professor Sigerist contributes an attractive preface. Dr. Licht, who is arranging for a series of translations of Galen, adds a life of Galen, translated from the standard work of Daniel Leclerc. The presentation of the book is excellent, but it cries aloud for an index.

CHARLES SINGER.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

W.H.O. and Birth Control

SIR,—At the meeting of the Programme and Budget Committee of the World Health Organization at Geneva last month the Norwegian delegate put forward a proposal to set up an expert committee to "examine and report on the health aspects of the population problem." Many of the Catholic countries' representatives spoke against this proposal, and two of them, Eire and Italy, said that several countries might withdraw from W.H.O. if it ever included birth control in its programme. Eventually the proposal was withdrawn without a vote and no action of any kind was taken.

This is a serious matter. One of the main criticisms was that over-population is an economic and not a health problem. Surely it cannot be denied that it is both? Over-population leads to epidemics, undernourishment, and chronic ill-health. Improved health measures, on the other hand, are likely themselves to lead to overcrowding in countries in which the expectation of life is low, since they prolong life and therefore increase the population. Although this problem is certainly economic and social in its origins and effects, it is also a serious health matter. To argue that methods of population control should be developed and encouraged is not to say that living standards should not be raised and food supplies increased. Both are necessary. These arguments are commonplace for medical men.

There were other criticisms of the Norwegian proposal which derived from an opposition to birth control in principle, and which therefore tended to lose some of their objective validity. The Austrian delegate even said that, the duty of the medical profession being to save life, not to prevent it, W.H.O. should not concern itself with a problem whose only solution seemed to be birth control.

The United Kingdom representative spoke only twice at this meeting, once to suggest that a session be adjourned and once to oppose a suggestion on balloting procedure. He gave no support to the Norwegian proposal. At its recent annual general meeting the Family Planning Association passed a resolution which "regrets that at the recent meeting of the Programme and Budget Committee of the World Health Organization the United Kingdom delegate (1) did not support the Norwegian proposal to set up an expert committee to examine and report on the health aspects of the population problem, and (2) gave no indication of the feeling of the great majority of the people of this country that birth control advice should be made available to those who need and desire it."

This is an affair of the greatest consequence. The population of India is increasing by four million a year and of the world by 20 million. Food resources are not keeping pace. Within a few decades a crisis will be upon us. The situation requires that we do more than acquiesce submissively in the embargo imposed by those whose religious sensitivities are offended.—I am, etc.,

Family Planning Association, London, S.W.1. DAVID PYKE.

Decomposition of Trichlorethylene

SIR,—I think it is worth pointing out the danger of decomposition of trichlorethylene left for some time in an untinted glass container. It is possible for this to occur in a Boyle's apparatus that is not often used. So little trichlorethylene need be given for each case that there may seem no necessity to change the liquid in the apparatus; but it is important to obey the instructions that are issued with "trilene" and replace the drug every few days. As an experiment, I recently left some trichlorethylene in an uncoloured glass bottle in the light for three weeks; the drug was kindly analysed by Imperial Chemical (Pharmaceuticals), Ltd., and it was found that appreciable decomposition had taken place, with the formation of hydrochloric acid and phosgene. The trichlorethylene bottle on the anaesthetic apparatus should be of tinted glass, and trilene that has become discoloured or colourless should never be used for anaesthesia.—I am, etc.,

Oxford. GORDON OSTLERE.

Antihistamine Drugs and Skin Eruptions

SIR,—I was much interested in the article on fatal phenobarbitone eruptions by Drs. I. B. Sneddon and A. W. D. Leishman (June 14, p. 1276). I wonder whether the condition may have been aggravated in Cases 1 and 2 by the administration of antihistamines. That "anthisan" can aggravate a drug eruption is shown by the following case.

A young woman consulted me last spring on account of paroxysmal rhinorrhoea and was prescribed "anthisan," 100 mg. three times a day. Two years previously she had sustained a burn of the right elbow region, where she developed a localized skin eruption following local treatment with a sulphonamide cream. The rash had cleared in about a month and there had been no recurrence. On the second day of systemic "anthisan" treatment she developed a fixed skin eruption of the right elbow region which was an exact replica of that produced by the local sulphonamide two years previously. It settled in one week on withdrawing the "anthisan."

—I am, etc.,

Leeds. G. W. LEWIS.

Pulmonary Embolism from Calf Veins

SIR,—It is with the diffidence befitting one whose surgery is minor in a minor field that I venture to intervene in my friend Mr. V. J. Downie's dialectic (June 14, p. 1303), provoked by Mr. McNeill Love's prophetic warning (April 26, p. 920) that any surgeon who fails to balance his patient's heels on a pillow is morally responsible for subsequent pulmonary embolism. Nevertheless, I think Mr. Love has the truth of it. In the phrase "unconscious recumbency," *unconscious,* which must include *completely relaxed,* is vital to the argument, and may be taken as "common form." The point at issue is, then, What precisely is meant by *recumbency*?

In the patient well and truly anaesthetized, and muscularly relaxed, stretched at full length flat on his back on the table, the surgeon intent on major surgery does not think of his patient's legs. He does not see them, for they are covered.

But in the resection of varicose veins of the leg, one or both legs are fully exposed to view, and in the patient stretched at full length the superficial veins of thighs and legs, even when only one leg carries varicose veins, stand out blue and engorged.

Furthermore, the curious who follow the patient back to bed—lying there flat on back and stretched at full length, till consciousness returns—will see the superficial veins in the unoperated leg, thus unbandaged and exposed to view, blue and engorged.

Tilting upward of the distal end of the table, or bed, puts a quick end to this engorgement: though doubtless Mr. Love's balancing of heels on pillow is equally effective.

I have opined that the engorgement was caused by the ilio-femoral vein's taut stretching across the pelvic brim in the supine, muscularly relaxed, unconscious patient.—I am, etc.,

London, W.1. H. M. HANSCHELL.

SIR,—I am very pleased to see this important subject discussed in the recent correspondence in the *British Medical Journal.* Calf vein thrombosis is extremely common and is important for two reasons. First, it is from thrombosis

which develops quietly in the calf veins that pulmonary emboli arise. Secondly, if the thrombosis is extensive and extends above the knee and a "white leg" ensues, then the patient who is lucky enough to escape with her life will be left with a lifelong severe venous stasis disability. Ultimately, as shown by Dr. Gunnar Bauer, of Sweden, these thrombosed deep channels recanalize, stripping the deep veins of their valves. Finally, fully 90% of these cases develop varicose ulcers within 20 years.

Virchow named three factors which were causative in intravenous thrombosis. These factors were venous stasis, damage to the intima of the vein wall, and alteration in the composition of the blood.

Venous stasis on its own does not cause thrombosis. Blood can be kept fluid *in vitro* for a prolonged period in a length of vein, provided the intima is undamaged. Damage to the intima of calf veins certainly causes intravenous clotting. Despite extensive fractures of tibia and fibula in young people, involving obvious vein damage, pulmonary embolism is a rare complication. Further, these cases are immobilized in thigh-length plaster casts. Venous stasis is thereby added to venous intimal damage. To explain the rarity of pulmonary emboli in these cases it seems logical to assume that the thrombosis is limited to the damaged stretch of vein wall, where it is firmly anchored.

It would appear to me that in cases that develop pulmonary emboli the major factor lies in an alteration of the composition of the blood. The blood in these subjects has acquired an increased tendency to spontaneous thrombosis. This tendency seems to be absent in the young, except in the pregnant, and appears from the age of about 35 onwards.

In this middle-aged group and upwards compression of the calf veins during muscular relaxation on a hard operating-table will cause slight intimal damage and venous stasis. This, however, is quite sufficient to fire off or catalyse the thrombotic process. The thrombosis is out of all proportion to the area of damage and stasis. The intimal damage is insufficient to anchor the extensive clot. The cardiac end of the clot spreads up into normal vein and is only anchored distally by a thread. A slight movement detaches this clot from its slender attachment and it is swept on by the venous stream to lodge in the pulmonary circulation. The point to emphasize is that the small intimal damage and venous stasis were only, as it were, the match which fired off this exaggerated thrombotic tendency of the blood. The difference between the young person and the older person is that in the young the thrombotic response is limited to the area of damaged intima. In the older group the clot extends well beyond the damaged intima to lie within normal vein.

The purpose of this letter is to remind us all that there are three partners in the thrombo-embolic partnership. I agree with Mr. McNeill Love (April 26, p. 920) that patients' calves should always be supported on the operating-table to prevent intimal damage and venous stasis. But if we could remove the increased tendency of the blood to clot in an exaggerated way as well I think pulmonary emboli and white leg would become very rare diseases.

Perhaps the anticoagulants should be used more frequently prophylactically when operating on the middle-aged group of patients. It would seem, however, that the gain in the reduction of thrombo-embolic disease would be more than offset by troublesome bleeding at the operation site.—I am, etc.,

Shoreham-by Sea. ANTHONY J. PARTRIDGE.

Mepacrine in Skin Disease

SIR,—The recent introduction of mepacrine in the treatment of lupus erythematosus has raised the question whether we should experience once more the occurrence of lichenoid dermatitis which was seen as a complication of malarial prophylaxis at the end of the war. Most of those who treat these patients and who use mepacrine must by now be familiar with this complication of an otherwise highly successful treatment.

Though there is a good deal of individual variation, Miller and I found in a series of 20 cases seen in Singapore in 1946 that lichenoid lesions never developed before the end of the second month of mepacrine prophylaxis. The same appears to be the case in the mepacrine treatment of lupus erythematosus. My own case first developed the complication after 10 weeks on 0.1 g. mepacrine daily.

It would appear reasonable to suggest that the drug should not be given for more than six to eight weeks in the first instance. At the end of this a marked improvement will have occurred in all those patients who are responding to the drug. After that it should be discontinued for two to three months, depending on the rate of excretion as judged by the colour of the patient's skin. The drug should probably not be given again when lichenoid lesions have appeared, and it should certainly be discontinued as soon as the first lesions are observed. The patient should be seen at frequent intervals during treatment so that the drug may be stopped at the earliest possible moment. Lesions are apt to progress for some time even after the drug has been discontinued and the discomfort occasioned by the lichenoid dermatitis may be very marked.

—I am, etc.,

London, W.1. W. G. TILLMAN.

Is Metallic Mercury Poisonous?

SIR,—While I cannot claim to be the "authoritative opinion" that Mr. R. S. Handley (May 24, p. 1131) would welcome, yet I would venture to suggest that on theoretical grounds the ingestion of metallic mercury, even in large doses, is harmless.

It is stated that the mercuric ion (Hg^{++}) is the responsible factor for the pharmacological and toxic effects of the drug. Mercury compounds vary in their toxicity according to the ease with which they liberate this ion. It would seem logical that this should depend upon the degree to which they ionize. If they ionize little, then their toxicity should be low. The acid salts ionize much more than other mercury compounds, and are more toxic.

With regard to the clinical thermometer, its average content of metallic mercury is 9 grains (0.6 g.). Most of this will not be ingested, for the patient usually spits most of it out. Even should he do so, the dose is small and it seems unlikely that any harm could come of it. The leakage which occurs from a Miller–Abbott tube contains often two or three ml. of mercury, but, even so, it enters low down, and the lack of ionization or alteration in the metal would militate against absorption. In support of this the *Encyclopaedia Britannica* (14th ed., **15**, 271) says: "So relatively inert is metallic mercury that a pound of it has been given without ill effect in cases of intestinal obstruction, which it was hoped to relieve by the weight of the metal."

I would submit that metallic mercury (as opposed to its compounds) is therapeutically inert when taken by mouth. Dover's patient certainly came to no harm, and was possibly (apart from the psychological effect) wasting his money!—I am, etc.,

Sanderstead, Surrey D. H. JUDSON.

Dermatologist's Protest

SIR,—May I protest at the use by physicians, some of them quite eminent, of the term "disseminated lupus" for widespread lupus erythematosus? Dermatologists are often derided for their terminology, but it is at least accurate. Lupus implies a tuberculous infection of the skin unless it is qualified by an adjective such as erythematosus. Continental dermatologists have in fact dropped the word lupus in lupus erythematosus and refer to it as "erythematodes" in order to avoid confusion. We appreciate the interest now being taken by general physicians in skin conditions, but we do ask them not to debase our terminology.—I am, etc.,

Leeds, 2. F. F. HELLIER.

Responsibility and Registration

SIR,—In the recent case of Manchester Regional Hospital Board *v.* Jones (*Times* Law Report, May 30) we read with regret the following: "He (Mr. Justice Oliver) expressed the view that they (the Regional Board) were asking for trouble in putting a weapon like pentothal within reach of a recently qualified doctor and expecting her to handle it with the necessary knowledge and experience." The important parts of this *obiter dictum* are the words "within reach," because they indicate a modern tendency to regard young doctors as only partially qualified. Immediately after registration we are all legally entitled to tie a ductus arteriosus, but

only a fool would attempt it—by which we mean that six years of training do not teach us all there is to know, but should give us some idea of our own limitations.—We are, etc.,

PETER H. SHERWOOD.
DENISE G. SHERWOOD.

Bristol.

Erythrocyte Sedimentation in Anaemia

SIR,—Dr. K. B. Rogers is to be congratulated on his careful experiments (May 24, p. 1109), which confirm the suggestion of Lehmann (1948) that cells of low mean corpuscular haemoglobin concentration (M.C.H.C.) have a lower specific gravity than normal cells, and also the findings of Poole and Summers (1952) that such anaemic cells sediment more slowly in both anaemic and normal plasma than do normal cells. But he is hardly justified in deducing that the reduced specific gravity is the cause of the slower sedimentation, or in inferring—by omission—that other factors are of little importance. Bunting (1939) showed that in sickle-cell anaemia rouleaux formation and sedimentation were almost abolished by increasing the sickling above 50% by exposure to carbon dioxide, and it seems likely that the anisocytosis and poikilocytosis of iron-deficiency anaemia interfere with rouleaux formation in similar fashion and are more important than reduced specific gravity in retarding sedimentation. If specific gravity were the essential factor, high E.S.R.s in symptomatic hypochromic anaemias would not be so commonplace, and it would be difficult to explain the normal E.S.R. one sees in some normochromic anaemias.

I should also like to comment on the following points: (*a*) Stokes's law refers to the unrestricted fall of a single small sphere in a viscous fluid and can hardly apply to the fall of many irregular and unequal aggregates of distorted red cells jostling one another within the confines of a glass tube. In addition, each falling aggregate displaces an equal volume of fluid upwards, thus affecting the fall of following aggregates; this interference with Stokes's law is best seen in the gross sedimentation of myelomatosis and other conditions with raised globulin, when the smaller aggregates are actually swept upwards by the displacement currents of the larger, and may continue up to the top of the tube.

(*b*) Dr. Rogers underquotes me. I suggested (1950) that "minor increases of the E.S.R. in anaemia are of *at least* the same significance as similar increases in normal blood," and continuing clinical observation convinces me that they are more so. Dr. Rogers's finding reinforces my opinion.

(*c*) The validity of "correction" charts is accepted by Dr. Rogers, but these charts are based upon artificial blood samples produced by manipulating normal or anaemic specimens, and their application to various forms of anaemia is difficult to understand.

(*d*) The slightly abnormal (up to 19 mm.) Westergren E.S.R.s in my Table III were associated with advanced phthisis, cancer, pregnancy, and infection, and yet Dr. Rogers insists that the increased sedimentation must be attributed to the accompanying anaemia, though I imagine he would agree that all these conditions are regularly associated with much higher E.S.R.s. Equally devoted to the doctrine of the need for correction, others happily correct E.S.R.s to zero or even minus figures.

Dr. Rogers's Case 2 neatly illustrates the whole problem. His patient has cancer of the colon with gross anaemia and a Wintrobe E.S.R. of 47, which the "correction" charts say should really be 5. He then argues that fault must be found somewhere, since the man is obviously ill and his E.S.R. should not be normal, and as the "correction" charts must be right he blames the hypochromic cells for having too low a specific gravity and for failing to sediment as Wintrobe and Hynes and Whitby say they should. I should like to suggest that the high E.S.R. is related only to the cancer and not to the anaemia, and also that Dr. Rogers could hardly have produced a better example of either the fallacy or the danger of "correction."—I am, etc.,

RICHARD TERRY.

London, E.C.1.

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More Liaison Needed

SIR,—As a consultant psychiatrist I would like to support wholeheartedly the letter of Dr. C. A. H. Watts (May 31, p. 1193). I, too, would like to see more liaison between the general practitioner and the psychiatrist, and suggest a start might be made on the following points:

1. There is the husband who telephones and says he wants a bed for his wife, having apparently been instructed to do so by his general practitioner, who certainly would not dream of adopting this procedure to get a patient into a general hospital.
2. There is the patient who turns up at an out-patient clinic with a "letter" stating, "? Neurosis. Your sincerely."
3. There is the patient who turns up with a very helpful letter from the psychiatric point of view, detailing family history, past psychiatric history of patient, etc., but omitting to mention that the patient suffers from epilepsy.
4. There is the patient who is handed an unsealed letter by the general practitioner to bring to the clinic, and, human nature being what it is, reads the general practitioner's views on the case.
5. Most important of all, and perhaps explaining why psychiatrists are a little cautious in putting their findings on paper, there is the out-patient or in-patient on leave, who is shown a report from the psychiatrist to the general practitioner. This leads to all sorts of complications, and is not prevented by marking the letter "Personal and Confidential."

It is not suggested that the above happen frequently, but their incidence is sufficient to warrant this letter.—I am, etc.,

G. M. WODDIS.

Nottingham.

Skin Lesions in Ulcerative Colitis

SIR,—There is no doubt that these ulcers vary with the progress of the disease, and anything that tends to improve the ulcerative colitis is reflected in the healing of the ulcers. Excision and grafting of the ulcers are well worth carrying out. There appears to be no specific organism associated with the ulceration, but they do seem to improve with ultraviolet light locally (E3 dose), and lotions containing heavy metals. A case where multiple ulcers of the face persisted, although the general condition improved, was successfully treated by excision and grafting of the ulcers, the largest ulcer being 2 in. (5 cm.) in diameter. I do not know the subsequent history, as the patient moved from London. No colectomy or ileostomy was performed.—I am, etc.,

CHARLES HEANLEY.

London, E.1.

Torsion of Fallopian Tube

SIR,—There must be many unusual events and combinations of events that inevitably escape being put on record when they point no diagnostic principle, and the association of uterine fibroids with torsion of a normal appendage has no doubt occurred several times before it was so well reported by Dr. P. G. Seed (May 31, p. 1176).

On May 12 I performed a multiple myomectomy operation for uterine fibroids, the largest being 7 in. (17.5 cm.) diameter, and I noted an old torsion of the left Fallopian tube and ovary. The ovary had passed anterior to the round ligament, the twist being through three-quarters of a circle, and a secondary blood supply had been obtained through adhesions to the parietal peritoneum. It was not necessary to remove the ovary, but the ovarian ligament and twisted isthmic end of the tube were severed in order to replace it.

The fact that the patient could recall no particular attack of lower left-sided pain suggests that torsion of a uterine appendage may often pass undiagnosed, and the successful outcome shows that temporization would have a real place in treatment if the diagnosis could be firmly established without operation.

—I am, etc.,

W. G. MILLS.

Birmingham.

Wandering Spleen

SIR,—I was interested to read Dr. C. L. Hall's account of a wandering spleen (May 3, p. 958), but feel that splenectomy should not be performed in a malarious area except in cases of absolute necessity or when the patient is in a position to leave the area for good after the operation.

Anyone who has unsuccessfully battled to save a simple case of malaria in an African who has no spleen will realize that splenectomy in such areas very frequently means a suspended death sentence. I feel that splenopexy would have proved a safer prevention of torsion.—I am, etc.,

Que Que, S. Rhodesia. J. M. WARD.

Bladder Paralysis

SIR,—In their most interesting account (May 31, p. 1172) of transient bladder paralysis occurring in natives of South Central Africa, Drs. M. Gelfand and E. E. Barnett unfortunately quote only one case. Although the cerebrospinal fluid was blood-stained, presumably from trauma, there were 48 leucocytes per c.mm. The red cell count of the fluid was only 848, and with a peripheral blood count of 5,000,000 red cells per c.mm. and a peripheral total white count of 16,200, such traumatic contamination should account for only about two cells per c.mm. in the fluid. Taking the upper limit of normal as 5 cells per c.mm., trauma does not appear to account for a pleocytosis of 48 cells. It would have been of considerable interest to know the type of white cell present and the results of lumbar puncture in other cases.—I am, etc.,

London, N.21. HARVEY ALDERTON.

Night Pain with Fractured Clavicle

SIR,—I have only recently seen Dr. J. B. Marshall's letter (January 19, p. 163) on night pain with fractured clavicle, and I am prompted to record this similar case which, by coincidence, I was called to see the day previous to reading his letter.

There was a three-day history in a 2-year-old boy of whining, disinclination to take his usual rest, and unwillingness to climb the staircase unaided—a feat which he had previously performed many times.

At midnight on the third night he awoke screaming, and would neither sit up nor allow anyone to sit him up. His mother thought also that his right arm was not being used.

I saw him at midday lying quietly on his bed. He was apyrexial and movement of his right arm was unhindered. There was no axillary lymphadenopathy. However, he cried when I sat him up. Suspecting a damaged right clavicle, I palpated the bone and found a very slight palpable deformity of the lateral one-third, accompanied by a violent verbal protest. *X*-ray confirmed a greenstick fracture.

There had been no known history of any injury, and his mother had called me in only after the acute nocturnal symptom of pain.

—I am, etc.,

Singapore. LESLIE J. LEVENE.

Treatment of Migraine with Oestrogen or Androgens

SIR,—In the *Medical Echo* of January under the title of a "Headache Clinic," Dr. Lippman, of San Francisco, describes his experience of the treatment of migraine by oestrogen and androgens. Since January, 1948, I have been treating cases by the same means, but exclusively by the oral preparations: chiefly stilboestrol and "ethisterone" in the case of females, and methyl testosterone for males. A rather rigid formula for the diagnosis of migraine has been employed, the aurae recognized being chiefly visual, occasionally aural, and very rarely psychic.

In estimating the results of treatment, the cases of "potential migraine" have been included: that is, cases which suffer from migraine only during periods of exceptional stress or anxiety and who recover completely when this has ceased. It is interesting to speculate whether the same mechanism operates in these cases as in allergic migraine. The doses employed have been small; usually 0.5 mg. stilboestrol and 10 mg. of ethisterone, arranged as a rule for women to have stilboestrol for 20–24 days after a period and ethisterone for four to six days before the following period, with no overlapping of the two preparations. Young men respond rapidly and effectively to daily doses of 5 mg. methyl testosterone alone. I have not found it necessary to give oestrogens in males. Up to the present it seems that middle-aged men should not receive this treatment. The results have been satisfactory: a few cases of frequently recurring attacks have gone four to six months without a relapse, and the physical and mental improvement in severe cases has been marked. Some cases of menopausal migraine do better on ethisterone alone.

The treatment followed the observation that women suffering from menopausal flushes and an aggravation of their migraine were relieved of both by stilboestrol. It has been noted since that two women suffering from migraine, and now also having attacks of asthma, were spontaneously improved after the second month of pregnancy.—I am, etc.,

London, E.2 L. J. MOIR.

Growth of the Foetal Head

SIR,—We have read with interest the paper by Dr. I. Macdonald (April 12, p. 798), and also the succeeding letter by Dr. Derk Crichton (April 26, p. 922). Though we cannot accept Dr. Macdonald's conclusions, we congratulate him on his paper, and hope that the work he has done may be integrated with that of others in an attempt to decide this very important matter of the rate of growth of the foetal head.

Dr. Macdonald has demonstrated that the size of the biparietal diameter in a newborn infant bears a direct relationship to the apparent gestation period of that infant, thus confirming the original work of Scammon and Calkins, except that the mean rate of growth described by Dr. Macdonald is less than half that claimed by Scammon and Calkins. We are not satisfied, however, that Dr. Macdonald's work supports the assumption that there is a significant growth in the biparietal diameter *between the 36th and 40th weeks of gestation.*

Josephs in 1949 found no evidence of growth in the biparietal diameter in the four weeks preceding delivery at term, his evidence consisting of repeated radiological estimates of the size of the head *in utero*, together with direct measurements by callipers of the same head three days after delivery. Over a large series of cases an accuracy of 0.02 in. (0.05 cm.) was claimed. The method of cephalometry, which is an entirely new one, has not yet been generally published, though it is available in the form of an M.D. thesis in the University of Durham Library. The difference between his conclusions and those of Dr. Macdonald may possibly be explained, at least in part, by a difference in the interpretation of the phrase "the last four weeks of pregnancy."

The weaknesses in Dr. Macdonald's refutation of Dr. Josephs's work are:

(1) The date of the last menstrual period is not necessarily an accurate guide to the date of conception. In some of the cases in Josephs's series apparent post-maturity occurred: the confinement took place 10 days or more after the expected date, as judged by the date of the last menstrual period, though without production of a genuinely post-mature infant. In these cases, when the first (radiological) measurement was made at a stage of 36 weeks' gestation, *estimated from the last menstrual period*, there *was* apparent growth, but it took place over an interval of five and a half weeks or more, from the (presumed) 36th week to 41½ or 42 weeks.

(2) Dr. Macdonald has measured each head only once, after delivery, and cannot prove that, in any individual case, growth of that head took place during the crucial phase of 36–40 weeks of gestation.

(3) He does not give any estimates of the standard errors of any of the statistical averages which he quotes, although it is apparent from his diagram that there are considerable individual variations which must affect the means of his groups.

(4) The foetal head does *not* "descend into the pelvis at a uniform rate" thus producing "a systematic difference between the measured and the true biparietal diameters." It is kind of Dr. Macdonald to find an excuse for Josephs's alleged error, but it really is regrettable that this fallacious statement is still being promoted. In some cases the head does engage fully in a primigravida at the 36th week and remain there till term, but in the majority of cases the position of the foetal head is not determined by the duration of pregnancy. It is determined by a variety of factors which include the attitude of the foetus, the tone of the uterus and abdominal wall, the length of time a patient remains recumbent before the abdominal examination, and the state of the bladder. The change in the position of the foetus which takes place when a recumbent woman stands up can be shown radiologically, and varies from full engagement to complete mobility above the brim. Anyway, the method of cephalometry employed by Josephs has the same degree of accuracy

wherever the foetal head lies: among the 189 cases quoted by Josephs were 16 breech presentations and three cases with a transverse lie.

(5) For some years, predictions of the course of labour in cases of doubt have been made by the radiologist and obstetrician based on radiological pelvicephalometry performed at the 36th week of gestation. These forecasts have proved right in the large majority of cases; if the head *had* grown after the measurement was made, the forecasts should have proved unduly optimistic.

Our view of the growth of the foetal head may be summarized in the general statement, of which the first half is already fully accepted: "From an early stage in pregnancy the rate of growth of the foetal head decreases as compared with the rate of growth of the body, and at some point in pregnancy approximating to the 36th week of gestation, the rate of growth of the head slows down so that in the four weeks preceding delivery at term the amount of growth is negligible." It is true that this statement makes nonsense of the practice of induction at 36 weeks for disproportion, but if anyone still does carry out this practice the beneficial results, if any, are gained not because the foetal head is smaller but because it is more readily moulded—perhaps at the expense of cerebral damage.

There is nothing biologically unsound in the assumption that the date of labour is ordained four weeks in advance, apart from the incidence of complications producing uterine irritability. We do not know the real cause of the onset of labour, but there is evidence that the placenta becomes increasingly less efficient during the last four weeks of a normal gestation: alteration in hormone production, as a result of increasing relative insufficiency of the placenta, may control both the onset of labour and the rate of growth of the foetal head.—We are, etc.,

H. CAMPBELL.
SYDNEY JOSEPHS.
LINTON SNAITH.

Newcastle-upon-Tyne.

Vascular Changes During Anaesthesia

SIR,—We have read with interest the paper by Mr. Ralph Shackman and Mr. I. G. Graber (June 14, p. 1284), in which certain vascular changes are attributed to general anaesthesia.

We note that skin and rectal blood flows are inferred from the temperature changes in these respective sites. For the temperature of a part to be an index of its blood flow, conditions of heat elimination and arterial blood temperature must be constant. We are doubtful whether these criteria were fulfilled in the observations mentioned in the paper. Since it is unusual for the operating theatre temperature to be less than 22° C. and the toe temperature remained constant at this figure for 20 minutes before anaesthesia commenced, we must conclude that the patients had not reached equilibrium with the thermal condition obtaining in an operating theatre. As it appears that the patients had recently suffered an increase in environmental temperature, peripheral vasodilatation and a consequent fall in deep body temperature are to be expected. Moreover, this transition prevents one from interpreting directly toe or rectal temperatures in terms of toe or rectal flows.

We should therefore like to know what thermal conditions obtained during these experiments and when transfer to the theatre occurred.—We are, etc.,

K. E. COOPER.
D. MCK. KERSLAKE.
R. F. MOTTRAM.

London, N.W.3.

Educating the Public about Cancer

SIR,—A well-informed public, seeking early diagnosis and treatment of malignant disease, appears to hold out the best prospect of diminishing the present terrible fatality rate from carcinoma. It does surprise me that the excellent little book entitled *Cancer: Where We Stand*, by Professor Sidney Russ, has not received wider publicity, both in the profession and amongst the laity. As Lord Horder says in his foreword, we should be censured if we allow a false delicacy to screen us from telling the public what is the real position in regard to this menace and from pointing out how it can help to achieve victory. He regards this presentation of the subject of cancer as one of the clearest and truest that he has yet read and thinks it will go far to enlist the help of the public in reaching a solution of this still baffling problem. With these views I most heartily agree, and I wish that something more might be done to focus attention on this imaginatively written and instructive small book.—I am, etc.,

London, S.E.5. GUY BOUSFIELD.

Employment for Over-sixty-fives

SIR,—The expectation of life is rising steadily. At the beginning of this century it was about 46. In 1942 it was over 64. I believe the latest available American figures make it 69. The balance of population is swinging steadily towards the aged group. In 1901 the over-sixties made up 4.7% of the population. According to the Beveridge Report it is estimated that in 1971 this figure will have risen to about 17%. The morbidity of the aged is about seven times as great as that of the population as a whole. When a workman in any job has reached the age of 65 his day's work has generally about used up his physical strength, and in the evening his inclination is to sit and rest. That is to say, hobbies and sidelines have in many cases been dropped, and if that man is suddenly retired from his work he degenerates mentally and physically, and quickly becomes a burden to his family, and, soon, an expense to the State. Amongst other things, he and his group make heavy demands on the much-needed hospital beds of the country.

It is quite clear, therefore, that if the country is to be run successfully we are faced with a serious and rapidly increasing problem, and it is our duty to set about meeting it in the best possible way. I think that the first thing to do is to abolish the 65 retirement age. Everyone will admit that there are many types of work at which a man is perfectly efficient long after age 65. Consider the watchmakers of Switzerland. Or take the case of a hospital physician-specialist. The loss of the accumulated skill and experience of such a man may be serious to the hospital he serves and to the community in which he has worked. And even in the heavy industries there are many places and jobs for the older men. The engineering firm of Rubery, Owen and Co., in Staffordshire, has a special workshop for older men. Last year, when I got particulars of this workshop from the firm, the youngest man in it was 69, and the oldest 84. The men start at 8.30 a.m., one hour later than the main works, work a 7½-hour day five days a week, and earn about £4 per week. The accounts of the workshop are kept separate from the general accounts of the firm, and they show a profit. Old men perhaps work more slowly, but as a rule their attention is not so easily distracted from their work, and consequently they are less liable to make mistakes. They take a real interest in the job on hand and forget to think of, and work according to, the weight of the pay packet. They are less liable to be carried away by Communistic ideas, or to work to rule, or go on strike, and so they do a real service to the country by putting a brake on the inflation spiral.

The suggestion to raise the age of retirement from 65 to 67 is to my mind childishly futile. The proper thing to do is to have a compulsory periodical medical check-up on the worker's health and efficiency, starting at an age much earlier than 65, perhaps at 50 or 55, retirement depending on the report from this check-up. In this way the unfit would be discovered and weeded out so that they would not be acting as a clog on the work, and in this way also room would be made for the oncoming younger groups. The check-up could be done once or twice yearly and could be made a duty of the medical inspectors of factories.

The second thing to do is to establish geriatric units in every group of hospitals so that the sick aged should have the opportunity of being rehabilitated as quickly as possible, returned to work, to their own homes, or to suitable welfare homes. As an ex-hospital surgeon I know that in a general hospital until recent years there were neither the time, the facilities, nor the skill to treat the disabilities of the aged. The excellence and efficiency of the work now being done in geriatric hospitals are at last becoming freely admitted. Being now connected with a geriatric hospital, I have come to the conclusion that for some considerable time to come it would be better for geriatric hospitals to be quite separate from general hospitals. When the General Medical Council comes round to making a course of geriatrics compulsory for every medical student before taking his final examination, and the Nursing Council does similarly with nurses, then the geriatric unit might become a department of every general hospital.

—I am, etc.,

Ballymena, Northern Ireland. J. ARMSTRONG.

Myositis Ossificans

SIR,—The use of decalcifying agents in this disease has been tried on several occasions without sustained benefit. The effects of parathormone administration, which Mr. A. P. Bertwistle suggests (June 21, p. 1357), were reported by Creveld and Soeters (*Amer. J. Dis. Child.*, 1941, **62**, 1000). After prolonged trial they concluded that no mobilization of calcium had occurred from the ectopic bone deposits as determined by *x*-ray examination. Further, the level of serum calcium, which rose initially with treatment, fell to normal readings despite increasing dosage. As your correspondent suggests, this title is inaccurate, insomuch as it is the fibrous layers that are affected and there is no evidence of inflammation. However, though more appropriate names have been suggested, the condition remains known as myositis ossificans progressiva, the name given to it by Dusch in 1868.—I am, etc.,

London, S.W.3. ROY H. MAUDSLEY.

College of General Practice

SIR,—Members of the General Practice Steering Committee (see *Journal*, December 29, 1951, p. 1582) are discussing the ways and means of establishing for general practitioners an organization with broad educational aims, the whole object of which is to maintain a high standard of general practice. These discussions are at an early stage at present; but the members of the steering committee are unanimous in thinking that general practice in Great Britain should be strengthened and supported by such a foundation.

Among other suggestions (see to-day's *Supplement*, p. 335) the proposal has been made that this foundation should be in the form of a college with central headquarters and regional branches. Members of the steering committee know, from a great volume of correspondence, that there is widespread interest in the formation of such a college: and at the same time they are well aware of the many difficulties which lie in the path of those who undertake a project of this importance. Nevertheless, they believe that the time is opportune and the ultimate prospects hopeful.

The work of the steering committee,* and of the organization to follow it, will necessarily involve expense. The committee is confident from the letters received that numbers of general practitioners and others—consultants, medical students, and many of the public—are so keenly interested in the future welfare of general practice that they will be glad to make some practical financial contributions (as gifts). We hope that the sum received by this means will be sufficient to enable us to complete our pilot inquiry, to issue our first report, and to write to all those doctors who are interested. Contributions for this purpose will be gratefully received by the secretary of the steering committee.—I am, etc.,

7 Mansfield Street, London, W.1. J. H HUNT, Hon. Secretary, General Practice Steering Committee.

POINTS FROM LETTERS

Medical Directory

The Editor of the *Medical Directory* writes: The accuracy of the *Directory* depends on the return of the annual schedule, which has recently been posted to members of the medical profession. Should the schedule have been mislaid I will gladly forward a duplicate upon request. The full name of the doctor should be sent for identification. Communications should be addressed to the Editor, *Medical Directory*, 104, Gloucester Place, London, W.1.

*The chairman of the General Practice Steering Committee is the Right Hon. Henry Willink, Q.C. The original members were Professor Ian Aird, Dr. G. O. Barber (Essex), Mr. John Beattie, Dr. J. H. Hunt (London), Sir Wilson Jameson, Professor James Mackintosh, Dr. J. MacLeod (Aberdeenshire) (resigned later for health reasons), Sir Heneage Ogilvie, Dr. F. M. Rose (Lancashire), and Dr. A. Talbot Rogers (Kent). The following practitioners were recently invited to join the committee: Dr. G. F. Abercrombie (London), Dr. D. M. Hughes (Carmarthen), Dr. R. J. F. H. Pinsent (Birmingham), Dr. R. Scott (Edinburgh), Dr. J. D. Simpson (Cambridge), and Dr. J. C. Young (Belfast).

Obituary

J. F. TAYLOR, M.D.

With the death of Dr. J. F. Taylor on June 16 at the age of 66, bereavement is widespread. Throughout his career, in all the many facets of his life, he made friends as other men pick up acquaintances. He will be greatly missed in the United University Club, of which he had been chairman, in the pavilion at Lords, at Woking Golf Club, and among freemasons. As chairman of the Sydenham Medical Dining Club, a position which marked the most exclusive and intimate esteem of his colleagues, he seems to be irreplaceable. His life was an example of loyalty and service to every institution to which he belonged.

After leaving Cambridge John Frank Taylor went as a clinical student to St. Thomas's Hospital, qualifying M.R.C.S., L.R.C.P. in 1911. He took the degrees of M.B., B.Chir. two years later and proceeded M.D. in 1919.

After holding house appointments he served in the R.A.M.C. in the Balkan theatre of the 1914–18 war as a pathologist under the late Professor L. S. Dudgeon. Afterwards he worked in the pathology department of St. Thomas's Hospital under the same teacher.

In 1928 he was appointed pathologist to St. George's Hospital. Here he made his professional home and here his qualities as a man and as a teacher were completely fulfilled and fully appreciated. His election to a "chair" was spontaneous and without university sanction, but generations of well-taught students and colleagues knew him affectionately as "The Prof." The critical judgment of undergraduates accepts only the best; for years he was officially president of the students' club and always their counsellor and friend.

We at St. George's knew his worth. His approach to pathological problems was direct and he gave invaluable practical help to his clinical colleagues. His teaching was forceful, and the solid bricks of basic principles were pointed with his special humour. On his retirement in 1947 he was appointed consultant pathologist to the hospital—the first ever to hold this office. Only last October he took the chair at the annual dinner of the medical school and gave us, as always, an example of his humorous and flexible art in after-dinner speaking.

Of later years the medico-legal aspect of his work had occupied much of his time. He played an important part in some famous criminal cases, but even the more humdrum or sordid aspects of sudden death were made instructive by the drama and pith of his teaching, and his lectures on forensic medicine were eagerly attended.

His shrewd wisdom and kindliness made him an admirable examiner. A colleague who examined with him in the Primary Fellowship tells me that no one put a candidate more quickly at his ease, but that no one was less likely to have the wool pulled over his eyes by a glib answer. Beneath his bonhomie was a firmness outspokenly intolerant of those whose folly he considered harmful. After a life full of hard work and play he was settling into retirement in his own county of Essex. Death was untimely: for us his friends in all walks of life it could not be otherwise. —M. F. N.

H. W. GRATTAN, C.B.E., D.S.O., M.R.C.S.

Colonel Henry William Grattan, who died in the Lister Hospital, Hitchin, on April 30 at the age of 80, was born on April 11, 1872, the son of Dr. M. H. Grattan, of Ongar, Essex, and was educated at St. Lawrence College, Ramsgate, and at the London Hospital, qualifying M.R.C.S., L.R.C.P. in 1894. He took the D.P.H. of the London Royal Colleges in 1904.

Grattan entered the Army Medical Service—later the R.A.M.C.—as a surgeon-lieutenant in 1895, and after serving at home and in Burma and India he accompanied the Royal Inniskilling Fusiliers to the war in South Africa in 1901–2. After that war he served in West Africa for a short time. Being interested in preventive medicine, he acted as assistant to the late Sir William Leishman at Millbank and was in charge of the Army anti-typhoid vaccine department from 1906 to 1910. Then he served in India again and during 1912–14 was in command of the enteric depot and bacteriological laboratory of the northern army in India. For his good work there he received an official letter of commendation and was made an Honorary Surgeon to the Viceroy and was awarded the Kaisar-i-Hind gold medal. In the war of 1914–18 he served with the Army in France, and rose first to be the A.D.M.S. of a division and then D.D.M.S. of IX Corps. He was three times mentioned in dispatches and was awarded the D.S.O. After some service in Germany in 1919 he became Deputy Director of Hygiene at the War Office, an appointment he held until his retirement in 1921, in which year he was appointed C.B.E. During the last period of his Army service Grattan organized a scheme of maternity and child welfare for the wives and children of men serving in the Army.

He did not retire to a life of idleness but became the medical officer of health of the Biggleswade urban and rural district, the Welwyn rural and garden city urban districts, and the Hatfield rural district. This appointment did not complete his activities, for in the last war he was chairman of the Bedford medical board and for 10 years was divisional surgeon of the Letchworth division of the St. John Ambulance Brigade. Among the papers he had written for the *R.A.M.C. Journal* was a useful one on medical organization, with special reference to the transport of wounded in open warfare.

In 1902 he married Mabel, the eldest daughter of Mr. L. Rice, C.I.E. His last few years in Letchworth, where he had been living since his retirement from the Army, were clouded over by the loss of his dearly loved wife, whom he had devotedly helped to nurse, thereby to some extent undermining his own health. He leaves a daughter, Mrs. F. G. Grandage, to whom we extend our sympathy. Grattan is mourned by all who knew him and the sterling uprightness of his character and the sincerity of his convictions as a Christian. He was indeed a typical gentleman of the old school. Would that there were more like him.—H. H. K.

W. S. V. STOCK, F.R.C.S.

We record with regret the death of Dr. W. S. V. Stock, at Porthill, Stoke-on-Trent, on May 30 at the age of 79. William Stuart Vernon Stock was educated at the University of Bristol, qualifying M.R.C.S., L.R.C.P. in 1898. He took the degrees of M.B., B.S. of the University of London in 1900 and M.B., B.Ch. of the University of Bristol in 1910, and became F.R.C.S. in 1907. After holding house appointments at Manchester Royal Infirmary, Bristol Royal Infirmary, and Bristol General Hospital, he became anaesthetist to the gynaecological department of Bristol Royal Infirmary and to the Bristol Eye Hospital, and, later, tutor in anaesthetics in the University of Bristol. He was also consulting anaesthetist to the Ministry of Pensions Hospital at Bath.

We are indebted to Professor R. S. S. Statham for the following appreciation: I had the privilege of knowing Dr. W. S. V. Stock for a period which began in 1910 and did not end until 1940, when I left Somerset to rejoin the R.A.M.C. During all that time I was in constant and close association with him, and a very deep and enduring friendship ensued. He was of the greatest assistance to me and to my colleagues in our early days, when I was a resident, and later a registrar, at the Bristol Royal Infirmary. We were closely associated in the gynaecological department, to which he was the anaesthetist. He was a first-rate mechanic and cabinet-maker, and designed and constructed many ingenious machines for the administration of various mixtures of anaesthetics. His great pleasure in the successful working of those ingenious bits of apparatus made it impossible to do anything but applaud, even if the depth of anaesthesia sometimes became rather light while the various adjustments were made. This mechanical ability led him to his major hobby, which was the building and racing of model yachts, at which he excelled, producing the most beautifully built and finished models. Perhaps Stock's most striking characteristic was a charming old-fashioned courtesy to everyone he met. It was a delight to go to his house and meet his family, to whom he was devoted. He was always interested in any new scientific work, the more so when it affected his own specialty. He was wholeheartedly devoted to freemasonry, in which he held many positions of eminence. A great gentleman, a devoted friend, and a willing helper of all young and struggling students and residents, he will be mourned by all who knew him as one of the last of the old B.R.I. school.

R. V. C. writes: Some of us came to know Stuart Stock only during the later years of his professional life and during his retirement. Right to the end he maintained a lively interest in all the changes and advances in anaesthesia. Sometimes we would have wished him to stick to well-tried and satisfactory measures, but his everlasting enthusiasm was always quite disarming. The induction of anaesthesia for the simplest of cases could be exasperatingly slow, but faced with a difficult problem he was usually superb. Professor Statham has spoken of his courtesy and friendliness, qualities which endeared him to so many of us. It is good to know that he was spared a long or a trying illness. Only a week or two before his death he was with us in Bristol for an afternoon. Then he was just the same as ever—cheery, charming, courteous, and inquisitive about everything going on in the theatre. So shall we remember him.

WILLIAM IBBOTSON, F.R.C.S.Ed.

Dr. Norman Meachen (Ringwood) writes: In the early years of the present century Ibbotson's piano-playing in his rooms at the old St. Pancras and Northern Dispensary, where he was then resident medical officer, was a musical treat. Many a time after seeing out-patients there I would climb the old, dark staircase, sink into a cosy chair, and listen to him spellbound. A few years later he very kindly opened my lecture-recital on "Medical Musicians" in the Barnes Hall of the Royal Society of Medicine, charming the audience by playing his own dramatic poem, "The Monk of Dunfermline," with consummate taste and skill. Had he devoted his life to music alone he would have been accorded a place among the world's great musicians, but, as Mr. S. E. Birdsall (June 21, p. 1359) justly remarks, he was that *in esse*.

Dr. ALBERT MAURICE AMSLER, who died at Hawkhurst, Kent, on June 11, in his 76th year, was born on January 6, 1877, and was educated at St. Paul's School and at St. Bartholomew's Hospital, qualifying M.R.C.S., L.R.C.P. in 1902. Three years later he took the degrees of M.B., B.S. of the University of London. After some clinical assistantships and locums he joined Dr. W. H. W. Attlee in partnership at Eton in 1906 and practised there until he retired in 1935. He was a physician to King Edward VII Hospital, Windsor, with charge of the obstetrical beds. He was also a member of the Eton College medical board. He was a sound general practitioner with a special flair for midwifery and, though sometimes blunt in manner, his patients were very fond of him. Apart from his profession, he was a remarkably versatile man. He had a host of interests and hobbies, and everything he undertook he seemed to do well. As a day boy at St. Paul's all his pocket-money came from the breeding and showing of bull-terriers, and in later years he judged them several times at Crufts. While a medical student he played rugby and water polo and boxed for Bart's, and at Eton he often judged in the College boxing and diving contests. He could raise plants and breed foreign birds when others failed, and many experts came to Eton to see his beautiful garden and his aviaries. His cabinet-making, too, was the envy of amateurs. A few weeks before his death he was awarded the Royal Horticultural Society's coveted Victoria Medal of Honour. He had previously won the Lyttel Lily cup of the society. In 1912 he married Victoria, daughter of Sir William Fairbank, of Windsor, and she, with his daughter and a grandson, survive him. Many will feel sad at his death, for he was a most interesting man, a good and kind doctor and friend, and a loyal colleague.—W. H. W. A.

Medico-Legal

DOCTOR'S ACTION AGAINST EX-PARTNERS FAILS

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

On February 13[1] judgment was given by Lord Guthrie in the Court of Session against Dr. Joseph Frank Sharp, of Cricklewood, in an action against Dr. Alexander M'Gregor and his wife, Dr. Anne M'Gregor, claiming £3,000.

Dr. Sharp entered into partnership with the M'Gregors on April 1, 1948, and the partnership was dissolved on July 3 of the same year. Dr. Sharp's case was that it was dissolved on the terms that he was to be paid £1,000 down followed by £3,000 at the rate of £1,000 a year by instalments spread over three years. On July 4 a written agreement was entered into by Dr. Sharp, drawn up by his solicitor, in which he was employed by the M'Gregors as assistant at £1,000 a year. Dr. Sharp maintained that this position was intended to be only nominal, and that the "salary" was really the instalments on the sale of his share in the partnership.

The M'Gregors' case was that Dr. Sharp had accepted £1,000 in full settlement on dissolution of the partnership, and that the service agreement meant what it said and the salary was supposed to be earned. On November 12, 1948, they gave him a month's notice determining the service agreement, as under the agreement they were entitled to do.

In his judgment Lord Guthrie said that he had considerable sympathy with Dr. Sharp over the dissolution of the partnership, but his wife had admitted they had made a bad bargain when the agreement was signed, and the law could not protect them from the consequences of their failure to look after their own interests. He was not satisfied that Dr. Sharp had not agreed to the terms of the document drawn up by his own solicitor, or was induced to sign it by any misrepresentation made to him by Dr. M'Gregor. The action therefore failed.

[1] *Glasgow Herald*, February 14.

Medical Notes in Parliament

Tuberculosis in Foreign Workers

On June 19 Dr. BARNET STROSS asked whether the Minister of Health was aware that persons coming from abroad to seek work in this country might be suffering from active respiratory tuberculosis, and what steps he proposed to take to exclude those who suffered from this infectious disease.

Mr. MACLEOD said he was considering again with other Ministers concerned whether further steps should be taken. There was a check on aliens, but it might not reveal the presence of the disease. Organized groups of European volunteer workers were subject to *x*-ray examination. The question of the remainder was under discussion. He would bear in mind cases from Eire.

Identity Cards

Dr. A. D. D. BROUGHTON on June 19 asked whether it was still necessary, since the abolition of identity cards, for medical practitioners to ascertain the identity numbers of patients applying for treatment as temporary residents.

Mr. MACLEOD replied that this was necessary. Dr. Broughton then inquired whether the insertion of identity numbers upon National Health Service forms did not impose an additional administrative difficulty on medical practitioners. He asked whether Mr. Crookshank had discussed the difficulty with representatives of the medical profession before or immediately after the abolition of identity cards.

Mr. MACLEOD could not agree that the difficulties were so serious. It was not true that the elaborate administrative card system behind the identity card system still had to be maintained. Mr. Crookshank had made it clear that the numbers would be retained for the National Health Service Act.

Cross-infection Among Children

Colonel M. LIPTON on June 19 asked if Mr. Macleod was aware that one in five child patients contracted an additional illness while in hospital, according to the report by Dr. J. W. D. Goodall recently published in the *Lancet* (April 19, p. 807), and what action he was taking to deal with this.

Mr. I. MACLEOD said he was aware of the article. On the advice of the Central Health Services Council his predecessor as Minister of Health had sent to hospital authorities last October a series of memoranda on different aspects of the prevention of cross-infection in hospitals. Hospital authorities, as their capital resources permitted, were improving old-fashioned children's wards. At least half the cross-infections were common colds. It was true that many hospital authorities were not carrying out to the full the instructions sent to them, and he thanked Colonel Lipton for the publicity he had given to the matter.

Unpunctual Doctors

Colonel M. LIPTON also asked on June 19 what action Mr. Macleod was taking to deal with the unpunctual attendance of doctors at hospital out-patient clinics.

Mr. MACLEOD replied that this was a matter for the hospital authorities concerned. Colonel LIPTON asked whether these doctors were not reasonably well paid. Ought they not to "clock-in" on time to ensure a humane and efficient out-patient service? Mr. MACLEOD said the time of patients was valuable, but the House should not forget how very valuable also was the time of consultants and specialists.

Mr. D. CHAPMAN asked Mr. Macleod to encourage hospitals to tighten their appointments systems so that out-patients attended at times suitable to the specialists. Mr. Macleod

said that was a matter for the hospital authorities. It was a long time since the Ministry had issued guidance on the matter, and he would consider whether it would be helpful to do so again.

Beds for Mentally Deficient Children

Mr. SOMERVILLE HASTINGS on June 19 asked what steps the Minister of Health was taking to provide further accommodation in institutions for mentally deficient children: there were approximately 4,000 cases waiting, half of whom were regarded as urgent, for vacancies in 7,120 beds.

Mr. I. MACLEOD said the attention of regional hospital boards had been drawn to the urgency of this matter. They had been asked to give special attention to schemes for increasing the accommodation for this type of case. About 200 extra beds would become available for schemes now in hand, but that was negligible against the size of the problem.

Treatment for Haemophilia

On June 19 Mr. P. F. REMNANT asked what inquiries had been made into the treatment for haemophilia devised by Dr. W. A. Timperley, Sheffield, and what steps Mr. Macleod had taken to assess the value of this treatment.

Mr. I. MACLEOD said this treatment had been investigated by the Medical Research Council on three separate occasions, care being taken to follow the technique recommended by Dr. Timperley. In every case the Council's investigators failed to find any evidence in support of the claims made. The treatment could be prescribed under the general medical service if a doctor happened to think it suitable. The fullest investigation had taken place into the claim, and in 1937 Dr. Timperley had been given a grant by the Medical Research Council.

Alcohol and Road Accidents

In the course of a debate on road safety on June 20 Mr. S. E. VIANT, the opener, referred to the risks to motorists of drinking. He said that 12 years ago a Select Committee had advised a scientific blood test. It was hardly an infringement of personal liberty to take two or three drops of blood from the lobe of an ear, which had to be set against blood spilt on the roads.

Later, Viscount HINCHINGBROOKE said a small glass of light sherry was not a bad stimulus to good driving; in fact it was a very good stimulus indeed. A very small quantity of any drug could do a lot to keep the senses keen.

Mr. SOMERVILLE HASTINGS pointed out that the effect of a drink could last for very long periods. It was for that reason that B.O.A.C. insisted that its flying crews should not only take no drink while working but should not drink anything for at least eight hours before going on duty. The British Medical Association in an inquiry a few years age found that 3 oz. of whisky (six tablespoonfuls) had a marked psychological effect in diminution of attention and control, and reduction in reasoning power. 90% of professional drivers employed by public authorities and private companies were forbidden to take drink when driving cars on the road. In Sweden, Denmark, Norway, Holland, Turkey, Germany, Switzerland, and parts of the United States the blood alcohol test was officially recognized as evidence. In some countries it was actually compulsory. In Denmark since the test was introduced the percentage of accidents due to drinking had gone down from about 10 to 2. However, the test was not infallible, because it had been shown recently, particularly by Professor Leonard Goldberg in Stockholm, that those who habitually took a lot of alcohol could take more than 0.15% in their blood without necessarily becoming public dangers. He thought that every police station should exhibit a notice saying that anyone charged with being under the influence of alcohol while in charge of a car could have a blood test if they asked for it. The notice should state clearly the value of the test not only in convicting but in proving innocence.

Mr. M. HIGGS said that the greater the penalty the more apprehensive magistrates became of convicting. They would not want to take away a lorry driver's driving licence if that took away his living.

Mr. CHUTER EDE said that the meaning of the word "drunk" had changed. When he was in the Army, if a man could take off his boots before he got into bed he was not drunk. He did not think blood tests were satisfactory, because they were not infallible. The same amount of alcohol, particularly a small amount, had very different effects on different people. Even in the countries where it was used the test was not yet regarded even by medical opinion as an infallible one. He would like to see it made possible for the motorist himself to ask for a blood test.

In replying to the debate Mr. G. BRAITHWAITE, for the Government, said he agreed completely with what Mr. Chuter Ede had said.

Tuberculosis in Singapore.—Over 500 beds are constantly in use for tuberculous patients in Singapore. It is hoped to start work in the near future on a new sanatorium which will provide a further 300 beds. The medical plan for Singapore envisages provision of 1,100 beds. There is a general shortage of trained staff and of staff accommodation.

Poison and Antidote.—The Poisons Board advises against legislation making it legally obligatory to have an antidote printed in label form on bottles, jars, or receptacles containing dangerous drugs, poisons, and the like.

Prescription Machines.—Twenty-two machines have been purchased by hospital committees for the purpose of collecting the shilling for prescriptions in out-patient departments; 237 machines have been hired.

Universities and Colleges

UNIVERSITY OF BIRMINGHAM

The Council of the University is to recommend to the next meeting of the Court of Governors that Professor H. F. Humphreys, Vice-Principal and Director of Dental Studies and Professor of Dental Surgery, be appointed Vice-Chancellor and Principal for one year from October 1, in succession to Sir Raymond Priestley, who retires on September 30. Professor Humphreys will resign his two appointments on September 30.

Professor A. P. Thomson, Professor of Therapeutics and Dean of the Faculty of Medicine, has been appointed Vice-Principal of the University for one year from October 1, in succession to Professor Humphreys. He will continue to be Dean of the Faculty of Medicine.

Honour schools in medical sciences will be established within the Faculty of Science as soon as the necessary facilities are available. The objects of their establishment are (*a*) to provide an adequate basic training at honours degree level for those intending to work in one or other of the scientific fields associated with medicine in universities, research institutions, schools, hospitals, and industry; and (*b*) to provide a one-year course for selected medical students leading to the same degree. The present honour school in anatomy and physiology will be replaced by (1) an honour school in anatomical studies, to be the responsibility of the professor of anatomy; (2) an honour school in physiological studies, to be the responsibility of the professor of physiology; (3) an honour school in medical biochemical studies, to be the responsibility of the professor of pharmacology.

Dr. J. W. Gerrard, Lecturer in Paediatrics and Child Health, has been appointed First Assistant in the department from April 1.

The title of Reader in Chemical Pathology (Cancer Research) has been conferred on D. L. Woodhouse, Ph.D., M.Sc., Lecturer and Senior Research Fellow.

Dr. Edward Park, of Johns Hopkins University, has accepted an invitation to be the first Leonard Parsons Lecturer and will deliver two lectures in 1953.

FACULTY OF OPHTHALMOLOGISTS

At the Council meeting of the Faculty, held on May 9, the following were elected officers for 1952–3: *President*, Mr. O. M. Duthie; *Vice-presidents*, Mr. R. C. Davenport, Mr. A. B. Nutt; *Honorary Secretary*, Mr. J. H. Doggart; *Honorary Treasurer*, Mr. E. G. Mackie.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending June 7 (No. 23) and corresponding week 1951.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1952 Eng. & Wales	1952 Lond.	1952 Scot.	1952 N. Ire.	1952 Eire	1951 Eng. & Wales	1951 Lond.	1951 Scot.	1951 N. Ire.	1951 Eire
Diphtheria	29	0	6	0		37	3	15	2	2
Dysentery	210	38	87	1	2	682	145	101	6	
Encephalitis, acute	7	1	1			0	0	0	0	
Enteric fever:										
Typhoid	5	0	0	0	1	7	0	0	1	0
Paratyphoid	13	1	1(A) 4(B)	0	1 (B)	15	1	0	0	
Food-poisoning	205	8		0		59	19		0	
Infective enteritis or diarrhoea under 2 years				11	25				12	16
Measles*	6,348	553	404	192	315	14,746	420	467	296	201
Meningococcal infection	23	1	16	1	5	53	1	17	2	
Ophthalmia neonatorum	36	2	7	0		40	0	11	1	
Pneumonia†	371	14	164	5	3	372	12	158	6	
Poliomyelitis, acute:										
Paralytic	42	7	8	3	1	32	2	4	0	
Non-paralytic	19	1				20	0			
Puerperal fever§	204	29	11	1	1	84	6	12	0	2
Scarlet fever	843	72	189	14	43	972	102	144	19	47
Tuberculosis:										
Respiratory			136	32				147	67	
Non-respiratory			27	8				28	15	
Whooping-cough	2,075	112	173	74	90	3.275	207	351	63	39

DEATHS in Great Towns	1952 Eng. & Wales	1952 Lond.	1952 Scot.	1952 N. Ire.	1952 Eire	1951 Eng. & Wales	1951 Lond.	1951 Scot.	1951 N. Ire	1951 Eire
Diphtheria	0	0		0	0	*0*	0	*0*	0	0
Dysentery	0	0		0			0		0	
Encephalitis, acute		0			0		0			0
Enteric fever	0	0		0		*0*	0	*0*	0	
Infective enteritis or diarrhoea under 2 years	12		1	2	0	*8*		*3*	4	0
Influenza	3	0	2	0	0	*9*	3	*0*	0	0
Measles		0		0	0		0	*0*	0	1
Meningococcal infection		0					0	*0*		
Pneumonia	135	23		11	4	*139*	22		5	7
Poliomyelitis, acute	0	0			0	*4*	1			0
Scarlet fever		0		0	0		0	*0*	0	0
Tuberculosis:										
Respiratory	99	10	10	1	10	*111*	18	*25*	7	7
Non-respiratory		0	5	2	0		2	*2*	1	5
Whooping-cough	5	1		0	0	*0*	0	*0*	0	1
Deaths 0–1 year	206	24	21	12	9	*224*	32	*22*	12	11
Deaths (excluding stillbirths)	4,285	591	509	111	130	*4,378*	687	*578*	98	159
LIVE BIRTHS	6,966	1094	946	217	406	*7,358*	283	*986*	257	497
STILLBIRTHS	186	23	22			*186*	23	*17*		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Vital Statistics

Infectious Diseases

Infectious diseases were less prevalent in England and Wales during the week ended June 7, and falls were recorded in the number of notifications of whooping-cough 520, measles 268, scarlet fever 72, and dysentery 66.

Only small variations occurred in the local trends of scarlet fever. The decrease in incidence was mainly contributed by the south-eastern counties with 52 fewer notifications than in the preceding week. The largest decreases in the returns for whooping-cough were Lancashire 80 and Surrey 64. The largest fluctuations in the local trends of measles were decreases in Norfolk 137 and Lancashire 103, and rises in Gloucestershire 88 and Middlesex 73. The number of notifications of diphtheria was 5 more than in the preceding week; the largest return was Warwickshire 7 (Birmingham C.B. 6).

The number of notifications of paratyphoid fever was 32 less than in the previous week. Four cases were notified in Wales during the week, compared with 33 and 43 in the two preceding weeks.

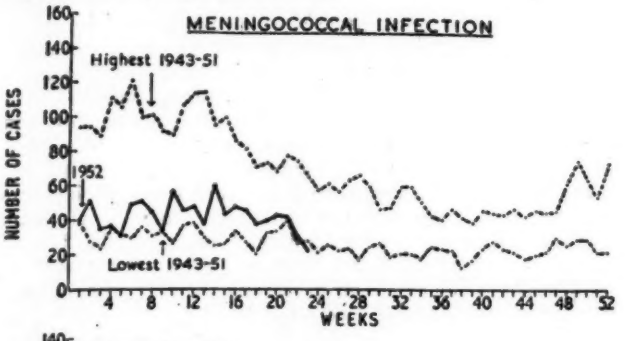
The number of notifications of dysentery was the lowest for 30 weeks. The chief centres of infection during the week were Lancashire 49 (Manchester C.B. 17, Oldham C.B. 12), London 38 (Islington 13), and Yorkshire West Riding 20.

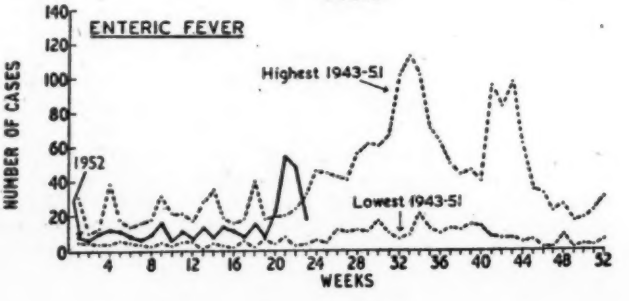
One hundred cases of food-poisoning were notified in Sheffield C.B. during the week.

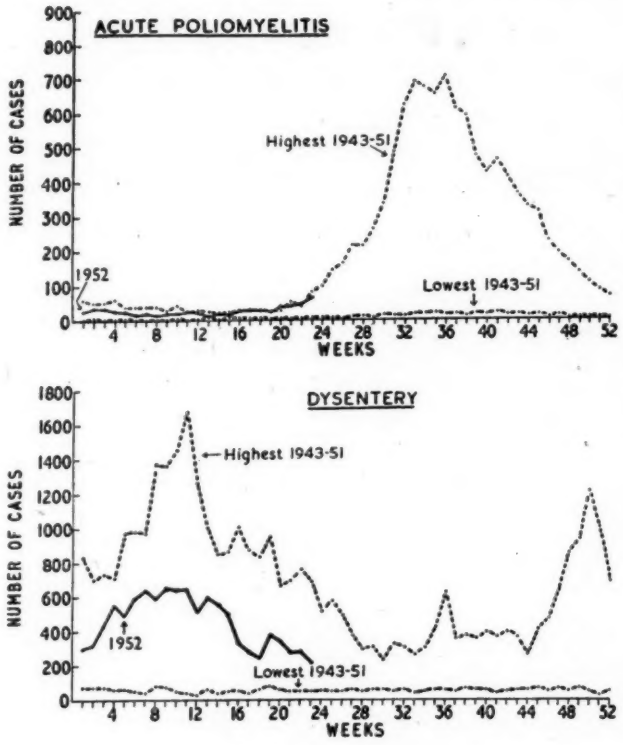
The number of notifications of acute poliomyelitis was the largest for 29 weeks. The paralytic cases were 16 more and the non-paralytic cases 1 more than in the preceding week. The largest returns were Durham 10 (Gateshead C.B. 6), Middlesex 8 (Harrow U.D. 6), Warwickshire 8 (Birmingham C.B. 7), London 8, and Yorkshire West Riding 6. The recent level of the incidence of acute poliomyelitis is above the level for the same period of last year and for the peak years of 1947 and 1949. This early rise of the seasonal trend does not necessarily mean that the summer will produce a large epidemic.

Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported during the nine years 1943–51 are shown thus - - - - - -, the figures for 1952 thus ———. Except for the curves showing notifications in 1952, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.







Week Ending June 14

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 845, whooping-cough 2,513, diphtheria 19, measles 7,307, acute pneumonia 427, acute poliomyelitis 48, dysentery 188, paratyphoid fever 32, and typhoid fever 4.

Medical News

July Family Doctor.—Number 7 of Volume 2 contains a wide range of articles grouped under such headings as Baby Features ("They need their Mothers" by John Bowlby, "Must Childbirth be Painful?" by Chassar Moir, "A Baby has Two Ends" by Dermod MacCarthy, "Is Baby Breathing?" by Frank Falkner), Home Section ("Making the Most of Fruits," "Summer Shopping," "Garden Parties," etc.), and Special Features ("The Strange Ways of Warts," "Blueprint for an Olympic Champion" by Sir Arthur Porritt, "Climbing Everest" by Raymond Greene, "Getting the Deaf into Circulation" by R. Scott Stevenson, etc.). This partial list displays the authority of the contributors and the variety of the subject-matter, which also includes in this issue stammering, athlete's heart, peptic ulcers, and worry. The aim is not only to give practical advice on health in the home and the upbringing of children but to show how medical men participate in all kinds of activities outside the consulting-room—for instance, in the Olympic Games ("Profile of Roger Bannister"). The presentation has greatly improved in attractiveness and polish in the last two issues.

Leverhulme Research Scholarships.—The council of the Royal College of Obstetricians and Gynaecologists is in a position to grant two scholarships of £500 a year each, renewable for a second and third year, for the purposes of research into problems connected with obstetrics and/or gynaecology. The scholarships may be held simultaneously with another appointment if, in the opinion of the council, sufficient time is available for research. Regulations governing these grants and forms of application can be obtained from the secretary, 58, Queen Anne Street, London, W.1.

More Money for Students.—Two thousand State scholarships will not merely be offered, but awarded, this year, which means that an extra 500 undergraduates will be supported from October, 1952, as compared with last year. Students whose parents' incomes range from £450 to £2,200 a year are eligible to receive financial aid, and, as the Minister of Education announced on June 20, the maximum rates have been increased from £265 to £288 a year for Oxford and Cambridge, £241 to £254 for London, and £215 to £230 for other universities. Some increases have also been made in the allowances for students' wives and children, and changes in the amounts allowed for the vacations.

Edinburgh University Club of London.—The Edinburgh University Club of London held its 246th dinner at the Savoy Hotel on June 18, with Mr. Scott Stevenson in the chair. It was not surprising that many of those present belonged to the medical profession, two of whom entertained the company with songs of Scotland, Mr. R. C. Farrow being at the piano and Mr. G. H. Macnab surprising the visitors with the excellence of his voice. Lord Horder was the guest of honour and proposed in a witty speech the health of the Club.

South-west Regional Paediatric Club.—The club met at Taunton on May 17 and 18. Cases were shown and discussed by Dr. J. Coates and others, and the following papers were read: "Mediterranean Anaemia," by Dr. A. H. Banton; "Twenty-five Years of Experience in Child Health," by Dr. Davidson; "Sexual Precocity," by Dr. H. R. Jolly.

Society for Relief of Widows and Orphans of Medical Men.—The annual general meeting was held on June 11, with the president, Sir Robert A. Young, in the chair. Membership of the Society at the present time is 235, and at the end of the year 34 widows were in receipt of grants. A by-law has been altered to enable widows whose yearly income exceeds £150 but is less than £200 to receive such grants as the court of directors shall determine. At the present time widows over 65 receive £75 and those younger £60 a year. During the year a present of £25 was made to each widow at Easter and again at Michaelmas. The total amount distributed was £4,484. The treasurer, Sir Ernest Rock Carling, reported that during the year income exceeded expenditure by £1,429, while £620 was received in legacies.

Homeless Children.—The International Children's Centre (Centre International de l'Enfance), which has its office in Paris at the Château de Longchamp, recently organized in London on June 9–18, in collaboration with the Home Office, the Ministry of Health, and the British Council, a colloquium on "Children Without a Family." The meeting was held at the British Council, with Mrs. Alva Myrdal, Director of the Social Sciences Department of Unesco, as president. After the opening by Mr. J. Ross, Assistant Under-Secretary of State, and Professor Alan Moncrieff, of the Institute of Child Health, experts invited by the International Children's Centre discussed such topics as adoption, family placing, institutional placing (children's homes or large orphanages), and the training of staff. Official representatives of 16 nations were present to compare the relevant laws in their respective countries and the efficacy of their different methods of dealing with the problem. The Social Affairs Division and the specialized organizations of the United Nations were also represented, together with observers from different British, Canadian, and French institutions.

38th Parallel Medical Society of Korea.—The society was formed on October 21, 1951, behind the lines in Korea at a United States mobile surgical hospital. The man mainly responsible for the formation and organization of the society was Lieutenant-Colonel William Yuckman, of the American Medical Corps, who is surgeon to the United States Third Division, and at the inaugural meeting he was elected the first president. Surgeons of other divisions were elected vice-presidents. Meetings of the society are held once a week at any one of three hospitals, all on the 38th parallel,

which means that attending medical officers actually cross the line and thus become eligible for membership. The roll of members now runs into several hundred, and the average attendance at each of the meetings is about 100. Recently a meeting was devoted to an account of the Commonwealth Medical Services in the field, with particular reference to an infantry division and to comparing our medical organization and tactics with those of the United States Army. Colonel G. Anderton, of the division, gave a general outline of the Commonwealth organization, which includes Australian, New Zealand, Indian, and Canadian contingents. Lieutenant-Colonel R. L. Marks described the organization and use of a field ambulance, and Lieutenant-Colonel A. G. Rangaraj spoke about the parachute field ambulance, an all-Indian unit of which India can be proud, for they have already parachuted into action with United Nations troops. Nothing but praise is heard about their turnout and bearing in Korea.

Plaque to Professor Stewart.—Professor H. R. Dean, Master of Trinity Hall, Cambridge, on June 12, unveiled a bronze plaque in honour of Professor Matthew Stewart, former professor of pathology at Leeds (*Yorkshire Post*, June 13). The residue of the money raised by subscription for the plaque, nearly £2,000, will be used to found a Matthew Stewart Lecture at Leeds University.

NEW ISSUES

Annals of the Rheumatic Diseases.—The next issue of this journal (Vol. 11, No. 2) will be available in a week's time. The principal contents include:

SHEEP CELL AGGLUTINATION TEST FOR RHEUMATOID ARTHRITIS. J. Ball.
INTRA-ARTICULAR HYDROCORTISONE (COMPOUND F) ACETATE. C. R. Stevenson, J. Zuckner, and R. H. Freyberg.
CORTISONE-ASCORBIC ACID INTERACTION AND THE PATHOGENESIS OF AMYLOIDOSIS. Gunnar Teilum.
INTERPRETATION OF MULTIPLE BIOPSIES OF SYNOVIAL TISSUE IN RHEUMATIC DISEASES. B. Cruickshank.
JOINT AND NEUROMUSCULAR MANIFESTATIONS OF PERIARTERITIS NODOSA. Edward W. Lowman.
PERIOSTITIS DEFORMANS. M. Soriano.
ANAEMIA OF RHEUMATOID ARTHRITIS. M. R. Jeffrey.
CORTISONE IN THE TREATMENT OF DEGENERATIVE JOINT DISEASE OF THE HIP. William F. Dowdell, Walter M. Solomon, Robert M. Stecher, and Ralph Wolpaw.

Issued quarterly; annual subscription £2 2s., single copy 12s. 6d.

Thorax.—The next issue of this journal (Vol. 7, No. 2) is now available. The principal contents include:

TERTIARY SYPHILIS OF THE LUNG AND ITS DIAGNOSIS. A. D. Morgan, W. E. Lloyd, and Sir Clement Price-Thomas.
FOUR CASES OF DIABETES INSIPIDUS AND PULMONARY DISEASE. John D. Spillane.
HUMIDIFICATION OF THE AIR PASSAGES. V. E. Negus.
MASSIVE HAEMOPTYSIS FROM ARTERIO-BRONCHIAL FISTULA IN PULMONARY TUBERCULOSIS. M. Berard and Jaubert de Beaujeu.
THE SO-CALLED MIDDLE LOBE SYNDROME. Bjarne Fretheim.
GASTRO-OESOPHAGEAL HAEMORRHAGE IN HEPATIC CIRRHOSIS. H. Butler.
PEPTIC ULCERATION AND PERFORATION OF THE STOMACH AFTER OESOPHAGECTOMY. V. S. Brookes and J. L. Stafford.
A RADIOGRAPHIC TECHNIQUE FOR THE STUDY OF LUNG PATHOLOGY. George J. Cunningham and John W. Miller.
MEDIASTINAL HAEMANGIOMA. O. Peräsalo.
A CASE OF PULMONARY CRYPTOCOCCOSIS. D. Barron Cruickshank and G. Kent Harrison.
THE FAILED THORACOPLASTY. R. Grenville-Mathers and H. J. Trenchard.
A' TABLE FOR PULMONARY RESECTION IN THE PRONE POSITION. Hans Rahbek Sørensen.

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SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday, June 30

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—5 p.m., "*The Long Pull in Cancer Research*," Imperial Cancer Research Fund Lecture by Dr. Peyton Rous, F.R.S.

Tuesday, July 1

HORSE SHOE CLUB.—At Medical Society of London, 11, Chandos Street, W., 5 p.m., annual general meeting.
KING'S COLLEGE HOSPITAL MEDICAL SCHOOL, Denmark Hill, London, S.E.—At Medical School Lecture Theatre, 4.30 p.m., "*Progress in the Treatment of Breast Cancer*," First Dorothy Platt Memorial Lecture by Professor D. W. Smithers.
●MIDDLESEX COUNTY MEDICAL SOCIETY.—At Middlesex Guildhall, London, S.W., 3.30 p.m., John Tate Meeting; 5 p.m., "*Observations on Pain and its Relief*," John Tate Memorial Lecture by Professor Lambert Rogers.
ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—5 p.m., "*The Effects of War on Surgical Practice*," Robert Jones Lecture by Sir Max Page.

Wednesday, July 2

YORKSHIRE SOCIETY OF ANAESTHETISTS.—At Leeds General Infirmary, 8 p.m., "*Some Reflections on Twenty-nine Years of Anaesthesia*," by Dr. J. K. Hasler.

Thursday, July 3

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, S.W.—5 p.m., "*The Puzzle for Therapy in Fluoroacetate Poisoning*," Bertram Louis Abrahams Lecture by Sir Rudolph Peters, F.R.S.
ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—4.45 p.m., "*The Evolution of Modern Tendon Surgery*," Moynihan Lecture by Dr. L. Mayer; 6 p.m., "*The Physiology of the Nasal Blood Vessels*," otolaryngology lecture by Professor D. Slome.

Friday, July 4

INSTITUTE OF CHILD HEALTH, Hospital for Sick Children, Great Ormond Street, London, W.C.—5 p.m., "*The Prophylactic Effect of Vitamin K in Newborn Infants*," by Professor P. Plum (Copenhagen).
INSTITUTE OF LARYNGOLOGY AND OTOLOGY, Gray's Inn Road, London, W.C.—5 p.m., "*The Prophylaxis and Treatment of Upper Respiratory Infections*," by Professor T. E. Walsh (Washington University).

Saturday, July 5

BRITISH ASSOCIATION OF ALLERGISTS.—At Biochemistry Lecture Theatre, University Museum, Parks Road, Oxford, 9.30 a.m., meeting. Papers will be read.

APPOINTMENTS

HOSPITAL FOR SICK CHILDREN, Great Ormond Street, London, W.C.—*Resident Assistant Physician*, R. G. Welch, M.D., M.R.C.P. *Assistant Medical Registrar*, C. H. M. Walker, M.D., M.R.C.P.Ed., D.C.H. *House Physician to Professor A. A. Moncrieff*, W. H. Cary, M.B., B.S., M.R.C.P., D.C.H.

SELLAR, MARY MORISON, M.B., Ch.B., D.P.H., Assistant Medical Officer of Health, County of Aberdeen.

SILVER, C. P., B.M., B.Ch., M.R.C.P., Assistant Tuberculosis Officer to Dispensary Clinic, London Chest Hospital.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Alban-Jones.—On June 11, 1952, to Delyth (formerly Lewys Lloyd), wife of Dr. D. J. A. Alban-Jones, a daughter.
Beale.—On June 19, 1952, at 13, Church Street, Bridgwater, Somerset, to Violet (formerly Pride), wife of Dr. J. Hanway Beale, a son.
Smillie.—On May 21, 1952, to Pamela, wife of Dr. T. G. Smillie, 195, Greenhill Avenue, Sheffield, a daughter.

MARRIAGES

Watson—Leach.—On May 17, 1952, at Halifax, Yorks, David Anthony Watson, B.A. M.R.C.V.S., of Wimbledon, London, S.W., to Barbara Leach, M.B., Ch.B., of Halifax.

DEATHS

Ascough.—On June 10, 1952, at King William's Town, South Africa, Matthew Thomas Ascough *M.C.*, M.R.C.S., L.R.C.P., D.P.H., late of Celyn, Romiley, Cheshire, aged 75.
Dunstan.—On June 21, 1952, at his home, Dunkeld, High Salvington, Worthing, Sussex, Walter Robert Dunstan, M.Sc., M.B., D.P.H., formerly of Lewes, Sussex, aged 75.
Turner.—On June 15, 1952, at his home, Albert Turner, M.D., J.P., of Fambridge Hall, White Notley, Witham, Essex.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Water Softeners and Lead Pipes

Q.—*I am told that if a water softener is fitted to a domestic water system with lead piping a form of chronic lead poisoning will result if the water is used for drinking or cooking. To prevent this it is apparently necessary to replace all lead piping by copper piping. Is this true?*

A.—The action of water on lead is a very complex subject. There are so many factors which can influence the result that it is often very difficult to give a general answer to a question upon it. In the present instance, however, the issue is more defined. A hard water is to be softened by the base-exchange process and the softened water is to be circulated through existing lead piping. The effect of base-exchange softening is to convert soap-destroying calcium and magnesium salts into the corresponding sodium salts which do not react with soap. Together with this, the *p*H value of the water is often raised slightly—i.e., the water becomes slightly alkaline. Neither of these changes results in an increase in the plumbo-solvency of the water, and there is no consequent need to consider the replacement of the lead piping by copper.

Intractable Urticaria

Q.—*What is the modern treatment, local and general, for severe and intractable cases of urticaria in adults?*

A.—The modern treatment of urticaria consists of both symptomatic treatment and treatment of the underlying cause. In the former the antihistaminic drugs are used, starting with small doses and increasing the dosage until symptomatic control is effected. It must be realized that side-effects vary considerably in individual patients, and different preparations may have to be tried before the most suitable is found. These drugs constitute the most important recent addition to our present symptomatic treatment. Adrenaline is still used in individual severe cases to bring a particular attack under control as rapidly as possible, while ephedrine is also occasionally used with an antihistaminic drug. Local treatment is less often necessary, but a few urticarial wheals in an individual are often best eased by the local application of an antihistaminic ointment, while more generalized wheals may be eased by warm alkaline baths, dusting the skin with talcum powder containing 0.5% menthol, or by a calamine lotion or cream containing menthol 1% and phenol 1%.

In considering the treatment of the underlying cause, it must be appreciated that urticaria is not a disease *per se*, but a skin reaction with widely different groups of causes—allergic, toxic, psychological, endocrine; not infrequently it is due to a combination of these factors. Intractable urticaria is usually an allergic manifestation in an allergic subject. Skin tests, however, have only a limited usefulness in this condition and trial elimination measures in the diet and the environment are essential. The allergic causes include (*a*) inhaled substances (pollens, feathers, animal hairs, house or occupational dusts, insecticides); (*b*) ingested substances, ordinary foods (egg, wheat, milk, potato, chocolate, fruits, fish, shell-fish), or common drugs (aspirin, quinine, phenolphthalein, sulphonamides); (*c*) injected substances such as an overdose of an allergen following serums, blood transfusion, the bites and stings of insects, or following injected drugs such as penicillin, liver extracts, or endocrine substances; (*d*) substances from bacteria (foci of infection in tonsils, teeth, sinuses), intestinal parasites or mycotic infection; (*e*) contact substances—the touch or lick of various animals or the use of their products in clothing or furnishings, the handling of certain foods (flour, fish) or plants (primula), the use of cosmetics, dyed clothes, or even adhesive plaster; (*f*) physical agents such as light, heat, cold, and trauma.

In chronic cases the first thing to do is to stop all previous medication, as drugs play such a large part in the causation of urticaria. Inhalant factors are a common cause: some cases are worse in bed during the night due to a sensitivity to feathers, down, or horsehair in bedding; other cases are confined to the summer months and are due to a pollen sensitivity; while in yet other cases the urticaria occurs during the day, when house and occupational dust play an important part—in these cases desensitization is necessary. Foods must also be carefully investigated by trial dieting, as results are so drammatically satisfactory when the cause is a food and this is eliminated from the diet.

Urticarial outbursts often follow emotional storms, anger or fright, or occur in states of fatigue. Such cases may be entirely psychological in origin and benefit considerably from psychotherapy and sedation; alternatively, such incidents may utilize available protective adrenaline and so render the sufferer more susceptible to some offending allergen acting during this adrenaline-depletion or exhaustion state. Premenstrual or menopausal urticaria will benefit by appropriate endocrine therapy. Foci of infection should be looked for and treated. Non-specific therapy such as histamine, autohaemotherapy, calamine, vitamin K, peptone, vaccines, and milk have all had their advocates and individual successes, but non-specific measures are becoming far less necessary as our knowledge of the various factors causing these illnesses increases.

Wax Baths

Q.—*I have a patient with advancing rheumatoid arthritis who is situated too remotely to profit by regular attendance at a physiotherapy clinic. Can you please advise me on the technique of giving wax baths?*

A.—When melted paraffin wax is applied to the skin it transmits heat by conduction. This causes local hyperaemia and sweating, but the sweat cannot evaporate owing to the impervious covering of wax and is therefore retained, causing further sweating until the wax cools. For therapeutic use it is customary to use paraffin wax, which melts at about 120° F. (49° C.), and to heat it in a double boiler. In the case of the hand this can be dipped directly into the wax two or three times until a satisfactory coat is formed. Wax can be applied to other parts with a brush. If it is left on for about 20 minutes the underlying skin is found to be hyperaemic, moist, and warm, and exercises should then be performed. Wax for this purpose, which is composed of about nine parts of commercial paraffin wax to one part of liquid paraffin, can be obtained from the commercial firms. Further details of the technique can be found on p. 382 of Kovac's *Electrotherapy and Light Therapy* (1945: Lea and Febiger, Philadelphia) or on p. 21 of Krusen's *Physical Medicine and Rehabilitation* (1951: W. B. Saunders, Philadelphia).

Scleritis

Q.—*What is the aetiology of scleritis?*

A.—Scleritis is more frequent in women than in men, and in them appears to have some relation to menstruation. Apart from pyogenic metastases, tuberculosis, leprosy, and syphilis, the aetiology is obscure. Most of the remaining cases represent an allergic reaction to some infection such as in the teeth, tonsils, etc.; many are gouty. Rarely the condition is met with in allergy to foods and epidemic parotitis.

Irradiation of Pituitary for Diabetes

Q.—*As experimental diabetes in dogs is benefited by hypophysectomy, is there any evidence to show that deep x-ray therapy to the pituitary gland is beneficial in cases of human diabetes, presumed to be of pituitary origin?*

A.—Deep *x*-ray therapy to the pituitary gland has been tried on a number of occasions in the treatment of human diabetes, but, except in those cases in which the diabetes has been secondary to a definite pituitary lesion, as in acromegaly and pituitary basophilism, the response has been negligible.

Beds in Sanatoria

Q.—*What is the recommended distance between tuberculous patients in general wards of sanatoria, and what distance is usually adopted in actual practice to-day? What type of mattress is found most suitable for sanatorium needs—rubber latex or interior sprung?*

A.—The recommended distance between bed centres for a fever hospital is 12 ft. (3.66 m.). Under present circumstances it is rarely possible to attain this standard in sanatoria, and 6 or 7 ft. is generally regarded as sufficient.

Either interior sprung or latex foam mattresses are suitable for sanatorium uses. The former are preferable, because they can be sterilized by steam under pressure, provided that they are kept flat. Latex foam mattresses can also be steam sterilized, but they deteriorate more quickly when subjected to this process.

Clinical Thermometry

Q.—*How do you explain an oral temperature which continues to rise over about 10 minutes? I have in mind a patient with pulmonary tuberculosis in whom, after the thermometer has been shaken down to 97° F. (36.1° C.), the temperature readings are: after two minutes 98.4° F. (36.9° C), after four minutes 98.6° F. (37° C.), and at the end of 10 minutes 99.2° F. (37.3° C.). Is this a common phenomenon, and what is this patient's "temperature"?*

A.—Oral temperature which appears to rise is a normal phenomenon, due to the delay in attaining a steady state within the mouth. In many practical physiology classes students read the mouth temperature at half-minute intervals, using clinical thermometers. A steady reading is frequently not obtained until the thermometer has been in the mouth for five minutes. If accurate readings of mouth temperature are required, five minutes under the tongue should be recommended rather than two minutes. This time can sometimes be reduced by keeping the thermometer under the tongue on one side of the mouth for half a minute, and then moving the thermometer across to the other side of the mouth, as suggested by Dr. Du Bois. The initial surface temperature may be many degrees below so-called body temperature in mouth-breathers, and the time to reach a steady state may be even longer than five minutes.

E.D.T. Crystals

Q.—*Is it dangerous to handle the E.D.T. synthetic crystals which are used in the telephone industry?*

A.—The difficulty of obtaining quartz crystals in recent years has led to the use of E.D.T. (ethylene diamine tartrate) to replace quartz in electrical circuits. No harmful effects have been reported from the handling of the finished crystals, which are chemically neutral and stable and are used in the form of slices or bars. Ethylene diamine, which is used in making the crystals, is known to cause irritation of mucous membranes. It affects particularly the eyes, nose, and pharynx, and may cause tightness in the chest. Dermatitis may also result from contact with the concentrated solution from which the E.D.T. crystals are grown. It is possible that dermatitis might occur from contact with finely produced E.D.T., but it is unlikely that the substance would be used in this form.

Bacteriology of Carious Teeth

Q.—*What is the common bacteriological flora of carious teeth? Are tubercle bacilli ever found?*

A.—The bacteria found most frequently in carious teeth are *Lactobacillus acidophilus* and *Streptococcus viridans*. It is believed that the enamel and dentine are decalcified by the acids released when these organisms ferment carbohydrates. Other organisms which have been reported include *Staphylococcus albus, Actinomyces, Neisseria catarrhalis* and *pharyngis, Proteus vulgaris*, yeasts, members of the *Bacillus* group, and *Clostridium welchii*. Tubercle bacilli have not been reported in carious teeth, but they have been found in a few cases of periapical infection in patients suffering from pulmonary tuberculosis.

Gräfenberg Ring

Q.—*How long should a silver ring be allowed to remain in the uterus?*

A.—Those who advise the Gräfenberg silver ring as a contraceptive (or more strictly as an abortifacient) say that it can be left in the uterus for a year, and sometimes longer, provided that the patient does not develop symptoms such as pain, menorrhagia, or discharge. It is emphasized, however, that in order to make sure of these points the patient must report to her medical attendant once a month. However, expert opinion in this country is opposed to a silver ring ever being inserted and left in the uterus. It involves a real risk of injury and infection as well as of menstrual disturbance, and it is not as reliable in preventing or disturbing implantation of the ovum as is sometimes supposed. The warning against the use of this instrument, published by the Family Planning Association in the *British Medical Journal* (1946, **2**, 599), still holds good.

Vaccination

Q.—*Where can I find information on the best way of vaccinating against smallpox?*

A.—On p. 139 of the book "*Any Questions?*" The book can be obtained for 7s. 6d. (postage 6d.) from the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1.

NOTES AND COMMENTS

Eczema of the Hand in a Child.—Dr. H. C. SEMON (London) writes: With reference to the question about a child with eczema of the hands ("Any Questions?" June 14, p. 1312), in a similar case, a girl of 7, the mother co-operated by listing in detail all the child's activities from dawn till dusk. Peeling oranges appeared to be an almost daily occupation, and it was eventually established by a patch test that the volatile oils of orange peel were the direct cause of the recurrent and intractable dermatitis. A temporary relapse some six months later was traced to contact with grapefruit pee' Both these citrus fruits and lemon juice could be taken by mouth without harm to the skin.

Correction.—Dr. K. B. ROGERS informs us that a column of figures was wrongly placed in Table II of his article in the *Journal* of May 24 (p. 1109). In the lower half of the Table under the heading "Westergren Sedimentation Rate" the column of figures which appears under "Corrected Rates (Hynes and Whitby)" should have appeared under "C.V. c.cm."

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

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INDEX TO VOLUME I FOR 1952

Names of contributors are printed in capitals. Subject entries for original articles, leading articles, annotations, and correspondence are indicated by the use of the symbols (O), (L), (A), and (C), respectively. Some subjects dealt with under various main headings in the *Journal*—e.g., "Annotations," "Any Questions?," "Leading Articles," etc.—are also set out under these headings.

A

ABBOTT, J. D., FERNANDO, H. V. J., GURLING, K., and MEADE, B. W.: Pulmonary aspergillosis following post-influenzal bronchopneumonia treated with antibiotics, 523 (O); 537 (L)
ABDALLA, H.: Emetine and scorpion stings, 614
ABELSON, D. *See* HARRISON, R. J., 303
Aberdeen University. *See* Universities: Aberdeen
Abortion, criminal, 915
— habitual, 427 (L)
— — further observations on prophylactic value of progesterone pellet implantation (P. M. F. Bishop and N. A. Richards), 244 (O); 428 (L); (correspondence), 437, 440, 604, 819, 923
— hormone levels in, 623
— medico-legal aspects (D. Teare), 915; 911 (A)
— repeated, significance and future outlook, 339
— — yaws as cause, 830
— septic, mortality rates, 889
— — treatment (C. W. F. Burnett), 886 (O); 911 (A)
— surgical, in tuberculosis, 1030
— therapeutic, 915
— — in acute heart failure; method of induction, 1260
ABRAHAMS, Sir Adolphe: Changing clinical aspects of pneumonia, 156
ABRAMSON, H. A. (Editor): *Somatic and Psychiatric Treatment of Asthma*, 1288
Abstracts: *Index Bibliographicus*, 1090
Abstracts of World Medicine, publication changes, 1204
Abstracts of World Surgery, Obstetrics, and Gynaecology, incorporated in *Abstracts of World Medicine*, 1204
Accident-proneness and the selection of bus drivers, 150 (A)
Accidents by falling from windows, 991
Accommodation in ophthalmoscopy, 1042
Acetomenaphthone, effect in hypoprothrombinaemia, 414
Acetylcholine shock therapy in psychiatry, 964 (A); 1082 (C)
Achalasia, hydrostatic bag for dilatation (H. Burge), 1245
Acidosis, treatment of, 1196
Acne during menstruation, treatment of, 1312
Acriflavine, sensitivity to, 1206
Acrocyanosis, treatment of, 396, 670
Acrodynia, infantile; psychiatric study of 6 cases (D. Leys and K. Cameron), 191 (O)
— treatment, 605
— — with "chloromycetin," 383, 605, 709
Acrylic lenses, intra-ocular, in treatment of cataract, 1121 (A)
— prosthesis, for hip reconstruction, 48
— replacement after segmental resection of bone (J. M. P. Clark and A. C. Bingold), 903 (O)

A.C.T.H.:
Agranulocytosis successfully treated with (F. D. Hart, D. G. Wraith, and E. J. B. Mansell), 1273 (O); 1292 (A)
Allergy and A.C.T.H. (G. W. Pickering), 1207 (O)
Cortisone and A.C.T.H. in treatment of blood disorders; preliminary report to Medical Research Council by panel on haematological applications of A.C.T.H. and cortisone, 1261 (O)
Effect on capillary resistance, 994
Effect of repeated injections upon bone marrow (G. Hudson, G. Herdan, and J. M. Yoffey), 999 (O); 1135 (C)
Emergency operation on patient under A.C.T.H. and cortisone therapy (A. Beck), 636; 1303 (C)
In delirium tremens, 614
In general practice, 102
In hypopituitarism (C. F. Rolland and J. D. Matthews), 1220 (O)
In infective hepatitis, 860 (L)
In leprosy, 601, 602
Limitations, 380
Response to, in rheumatoid arthritis (J. H. Kellgren and others), 997 (O); 1016 (A) (correction, 1144)
Therapy in ankylosing spondylitis (F. D. Hart), 188 (O)
— of Schönlein-Henoch (anaphylactoid) purpura (E. J. S. Woolley), 259
Untoward Reactions of Cortisone and A.C.T.H. (V. J. Derbes and T. E. Weiss), 1072
Use in rheumatic diseases in U.S.A., 342
Actinomyces israeli, sensitivity of, to antibiotics (L. P. Garrod), 1263 (O)
Actinomycosis, paralaryngeal in a child (D. F. Davies), 363
— rapid cure, 277
ADAMS, D. M.: Ethyl chloride for unconscious hysterics, 1191
ADDISON, Lord, memorial service for, 172
— tribute in House of Lords (parliamentary note), 334
Addison's disease and pregnancy (T. A. Richards), 421; 708 (C)
Aden, hospital facilities at (parliamentary note), 665
Adenoids and tonsils, fatal staphylococcal septicaemia following removal (E. J. G. Wallace and O. G. Jones), 1231
Adiposity, aetiology of, 726
Adjudication award, 697 (L), 749; 718, 771 (parliamentary notes); preparing the case, 263 (L); Working Party, 1200, 1235 (L)
Adoption of children; Jocasta complex in adoptive mothers, 817
Adrenaline: *Nor*adrenaline, adrenaline, and the human circulation (H. J. C. Swan), 1003 (O)
— L-*nor*Adrenaline as a vasoconstrictor, 106, 385, 546
— — by intravenous injection, 223

ADRENALS: *See also* A.C.T.H.; Addison's disease
Cortex: Capillary resistance and adrenocortical activity; further observations (H. N. Robson and J. J. R. Duthie), 994 (O)
Carcinoma, with endocrine manifestations; case (R. J. Harrison and D. Abelson), 303 (O); (correspondence), 655
Function at death measured by level of circulating eosinophils (P. B. Jennings), 1055; 1249 (C)
Hormones in pregnancy, 621
Symposium of Colston Research Society and University of Bristol, 865
Function in rheumatoid arthritis, 345
Phaeochromocytoma with negative benzodioxane test (H. P. Tulloh), 531 (O)

Adrenocorticotrophic hormone. *See* A.C.T.H.
Aero Medical Association: 23rd annual meeting, 1089
Africa: Annual clinical conference of Uganda Branch of B.M.A., 158; 820 (C)
— *Global Epidemiology: A Geography of Disease and Sanitation*, vol. 2: *Africa and the Adjacent Islands* (J. S. Simmons and others), 1010
Age, old. *See* Old age
Agene: Effect on vitamins in flour, 614
— Medical Research Council investigations (parliamentary note), 824
Agenized bread (parliamentary note), 334
Agranulocytosis caused by amidopyrine; an avoidable cause of death (G. Discombe), 1270 (O); 1292 (A)
— successfully treated with A.C.T.H. (F. D. Hart, D. G. Wraith, and E. J. B. Mansell), 1273 (O); 1292 (A)
Agriculture, poisonous chemicals in; arrangements for blood tests, 1014 (L); 1024; 1135 (C)
Agriculture (Poisonous Substances) Bill (parliamentary notes), 1035, 1199, 1252, 1360
AHERN, R. T., and ARDEN, G. P.: Intra-articular streptomycin in tuberculosis of the knee, 466 (O); (correspondence), 766, 974, 1194
AINSWORTH, Sir Ralph Bignell, obituary of, 331
Air embolism, pathogenesis of, 892
— — post-mortem diagnosis by radiography (J. D. Taylor), 890 (O)
— purification, 1259
AIRD, I.: Pilonidal sinus of axilla, 902 (O); 1356 (C)
Albuminuria in gout, 670
Alchemy, 637
Alcohol; effect in angina pectoris, 502

ALCOHOLISM:
Alcohol and road accidents, 47, 1413 (parliamentary note)
Antabuse therapy, indications and contraindications, 931
Booklet issued by Advisory Council on Alcoholism, 499
Drunkenness among car drivers; trial by magistrate and jury compared, 161
Sobering effect of laevulose, 1041
ALDERTON, H.: Bladder paralysis, 1408
ALDRIDGE, W. N., and DAVIES, D. R.: Determination of cholinesterase activity in human blood, 945 (O); 1015 (C) (correspondence), 1135
ALEXANDER, G. L.: Attempted suicide, 921
Alkalosis, hypokalaemic, fatal with tetany during liquorice and P.A.S. therapy (N. J. Roussak), 360 (O)
— in Cushing's syndrome, 1205
ALLAN, T. M.: Antistin in habitual abortion, 437
ALLEN, A. C.: *Kidney: Medical and Surgical Diseases*, 423
ALLEN, C.: Experiment in peacemaking, 652
ALLEN, G. V., knighthood conferred on, 44
ALLEN, Robert Hugh, obituary of, 1034

ALLERGY: *See also* Asthma
Allergy and A.C.T.H. (G. W. Pickering), 1207 (O)
Annual general meeting of the British Association of Allergists, 380
Etudes Cliniques, Expérimentales et Thérapeutiques sur l'Allergie (P. Vallery-Radot), 204
House dust as an allergen, 35 (L)
Mechanism of anaphylaxis and allergy (M. Rocha e Silva), 779 (O); 807 (A)
Schönlein-Henoch (anaphylactoid) purpura treated with A.C.T.H. (E. J. S. Woolley), 259
Sensitivity to oak, 1092
Serum reactions and serum sensitivity tests (L. J. M. Laurent and H. J. Parish), 1294
Serum Sickness (C. von Pirquet and B. Schick), 1118
Tissue reactions to protein sensitization (J. R. Squire), 1 (O); 35 (L)

"Allethrin," 543
ALLINSON, B. P.: Nature cure, 1031
ALLOTT, E. N.: Future use of cortisone and A.C.T.H. in general practice, 103
ALSTON, J. M.: Mental health research fund, 975
Aluminium and silicosis, 829, 1206
Amanita phalloides, poisoning due to, 593
AMBROSE, G.: Hypnosis and asthma, 326
— Stage hypnotism, 921
America: Medical mission to Latin America (T. M. Ling and others), 756
— *See also* United States of America
American Journal of Tropical Medicine, 55
American Medical Association: Annual convention to be televised, 1039
American Orthoptic Journal, 172
Amidone, hazards in use of, 1260
Amidopyrine agranulocytosis: an avoidable cause of death (G. Discombe), 1270 (O); 1292 (A)
— — successfully treated with A.C.T.H. (F. D. Hart, D. G. Wraith, and E. J. B. Mansell), 1273 (O); 1292 (A)
Amine oxidase, 861 (A)
— — at sympathetic nerve endings, 784

p-AMINOSALICYLIC ACID:
Acquired sensitivity to (S. J. Steel), 415 (O)
Alkalosis, hypokalaemic, fatal, with tetany during liquorice and P.A.S. therapy (N. J. Roussak), 360 (O)
Hepatitis, toxic, during administration (A. G. Fergusson, J. P. McIntyre, and A. R. Gemmell). 855
In combined chemotherapy of streptomycin-resistant pulmonary tuberculosis, 1157 (O); 1179 (L)
In tuberculous meningitis (S. R. Jamieson), 83 (O); (corrections 165, 223), 329 (C)
Toxic effects, 58
Tuberculosis, pulmonary; chemotherapy in young adults; analysis of combined results of three Medical Research Council trials (M. Daniels and A. B. Hill), 1162 (O); 1179 (L)

AMSLER, Albert Maurice, obituary of, 1412
AMULREE, Lord: *Adding Life to Years*, 364
Amyloidosis and thrombocytopenic, purpura, in Hodgkin's disease (R. C. Gledhill and A. J. Shillitoe), 1337

ANAEMIA:
Cooley's anaemia, inheritance of, 880
Correction of E.S.R.; experimental study based on interchange of cells and plasma between normal and anaemic subjects (J. C. F. Poole and G. A. C. Summers), 353 (O); 369 (A)

ANAEMIA (*continued*):
Diagnostic test for use in general practice, 600
Haemolytic, associated with reticuloses (D. R. C. Willcox), 1322 (O)
— effect of A.C.T.H. and cortisone, 1261
— splenogram in, 989
In pregnancy, 476 (A)
Intestinal macrocytic anaemia (G. M. Watson and L. J. Witts), 13 (O)
Iron therapy, 407, 476 (A), 599, 817
Megaloblastic anaemias of pregnancy; response to animal protein factor; preliminary report (H. Foy, A. Kondi, and A. Hargreaves), 852 (O)
— in Indians, 220, 764
— in young adults; aetiological importance of occult idiopathic steatorrhoea (H. Conway), 1098 (O)
— of infancy (A. G. Rickards), 1226 (O)
Of hypopituitarism (V. K. Summers), 787 (O)
Pernicious, association with gastric carcinoma, 288
"Physiological," of pregnancy; effect of iron therapy (Nancy Benstead and G. W. Theobald), 407 (O); 476 (A); (correspondence), 599, 817
Severe, exchange transfusion in (T. Ward), 631 (O)

ANAESTHESIA: *See also* Analgesia
Dangerous drugs in the anaesthetic room; legal and practical requirements for recording, 1365
Dangerous hypotension after, 613
Death under anaesthesia, 715
English Pioneers of Anaesthesia (*Beddoes, Davy, and Hickman*), by F. F. Cartwright (C. Singer) (Nova et Vetera), 706
Explosion during anaesthetic (medico-legal note), 168; (correspondence), 273, 820, 870, 972, 1083
Faculty of Anaesthetists, 168; fourth annual general meeting, 825
For minor procedures (W. W. Mushin), 431; 711 (C)
Mode of Action of Anaesthetics (T. A. B. Harris), 423
Modified endotracheal connector, 1355
Reciprocal changes in blood flow of the limbs and rectum during general anaesthesia (R. Shackman and I. G. Graber), 1284 (O); 1409 (C)
Scottish Society of Anaesthetists, 1309
Syringe service for a department of anaesthetics T. H. S. Burns), 1127
T-piece technique for general inhalational anaesthesia; safe method of reducing operative bleeding (J. Bullough), 28 (O); 327 (C), 384 (C), 546 (C), 654 (C)

ANALGESIA:
Aid to analgesia of larynx and trachea (T. Dinsdale) (preparations and appliances), 707
Analgesics for the nose, 285
Portability of Minnitt apparatus, 328
Procaine, intravenous, for pain relief, 266 (A)
Trichlorethylene in labour, 108
Urethral instrumentation, 395
Xylocaine, 211 (A); 328, 488
— fatal injection, 280; 714 (C)

Anaphylaxis and allergy, mechanism of (M. Rocha e Silva), 779 (O); 807 (A)
— Serum reactions and serum sensitivity tests (L. J. M. Laurent and H. J. Parish), 1294

ANATOMY:
Anatomy Act, revision suggested, 544, 651
Anatomy in Surgery (P. Thorek), 639
Angewandte und topographische Anatomie (G. Töndury), 696
Applied Anatomy for Nurses (E. J. Bocock and R. W. Haines), 424
Comparative: *The Chordates* (H. W. Rand), 91
— *Anatomy of the Gorilla* (W. B. Atkinson and others), 365

ANDERSON, G. W. *See* SIMMONS, J. S., 1010
ANDRESEN, P. H.: *Human Blood Groups Utilized in Disputed Paternity Cases and Criminal Proceedings*, 1395
Andrewes, C. H., awarded Stewart Prize, 1184 (A)
ANDREWS, M. C.: What doctors prescribe, 486
— Damnable heresy? 1196
Androgens, influence on skeletal growth, 726
Angina pectoris attacks precipitated by cold, 118, 558
— — effect of alcohol in, 502
— — in medical practitioners, 509
Angioma venosum racemosum with angiomatous lesions of skin and omentum (I. Gilbert), 468 (O)
Anglo-French medical exchange bursaries, 555, 1309
Anhidrosis, thermogenic, 1346
Animal protein factor, response of megaloblastic anaemias of pregnancy to, 852
Annals of Physical Medicine, 445

ANNOTATIONS:
Abortion, septic, 911
Acetylcholine treatment in psychiatry, 964, 1082
Amidopyrine, danger of, 1292
Amine oxidase, 861
Anaemia in pregnancy, 476
Anaphylaxis, mechanism of, 807
Arteriotomy, 965
Bacteria at the sea bottom, 1343
Bacterial products against cancer, 429
Beginning to breathe, 97
Bevan versus Spens, 1291
Bladder tumours in dyestuff workers, 1123
Bonnier Scotland, 808
Brain tumours, location with isotopes, 428; 549 (C)

ANNOTATIONS (*continued*):
B.M.A.: Annual meeting, Dublin; scientific exhibition, 371, 379
— Film catalogue, 152
Bus drivers, selection of, 150
Cerebrospinal fever, epidemiology, 211
Chemotherapy, allergy, and tuberculosis, 702
Children, growth and development of, 963; 1133 (C)
Ciba Foundation Report for 1951, 592
Contraceptives by mouth? 1183
Co-operation in the Health Service, 912
Cortisone, responsiveness to, 1016 (correction, 1144)
Death on the table, 1181
Diffusion respiration, 430; 1358 (C)
E.S.R. in anaemia, 369
Family Doctor, 39
Family research at Peckham, 317; 490 (C)
Femoral neck nailing, 592
Fire extinguishers, poison in, 1122
Funnel chest, 370; 715 (C)
Great Ormond Street centenary, 371, 1184
Hair, physiology of, 369
Haiti experiment, 96
Health visitors, 1077; 1355 (C)
Honour the surgeon, 1293
Honours List, 1293
Hospitals: Closer link with the G.P., 151
House appointments, purpose of, 914
Hunger and hypothalamus, 752
Illegitimate children, 265
Income tax and Spens, 913
Infants of doubtful sex, 1292
Inflammation, 1123
Influenza, epidemiology, 209
Insecticides: Laboratory tests for agricultural workers, 1014
Intra-ocular acrylic lenses, 1121
Intravenous feeding with fat emulsions, 1400
Korea, epidemic haemorrhagic disease in, 477
Language disability, specific, 36; 325 (C)
Left hand, right hand, 591; 763 (C)
Libel and slander (Defamation (Amendment) Bill), 266
Lymphocytosis, infectious, acute, 96
"M. & B.I.," 98
Malaria: A new antimalarial, 1075
— prophylaxis and treatment 590; 1134 (C)
Medical care; "the easy approach," 1343
Mercury for children, 370
Motor-cycle accidents, 1183
Mumps, immunity in, 912
Muscle relaxants; brief flaccidity, 1342
Mycology: Organization for medical mycology, 1400
Nasal bleeding, 1124; 1197 (C)
National Formulary and "Prescribers' Notes," 701; 924 (C)
Nutrition in New Guinea, 37
On giving evidence, 644
Pantothenic acid, 643
Penicillin in the lungs, 1238
Petrol tax, 644
Photography: For those that have eyes, 1293
Photosensitivity and porphyrins, 477
Pneumoconiosis in gold mines, 965
Pneumonia, choice of antibiotic for, 37
Prescription charges, 1181
President, 1953–4, 1181
"Priscol" in cerebral arteriosclerosis, 98; 106 (C)
Procaine, intravenous, for pain relief, 266
Pseudocyesis, 538; 637 (C), 816 (C)
Pulmonary embolism, 809
Respiratory function tests, 95
Retirement, compulsory, from hospital service, 38
Rift Valley fever in South Africa, 267
Ross Institute Industrial Advisory Committee, 479
Royal Society: New Fellows, 703
Salmonella minnesota infection, 38; 53 (vital statistics)
Scarlet fever, epidemiology, 317
School medical service, 210; 490 (C)
School of Hygiene, Athens, 808
Science in Piccadilly, 1182
Sickness studies from insurance statistics, 753
Sleeping posture in phthisis, 316
Splenic puncture, 1016
Steroid metabolism in idiopathic hirsutism, 39
Stewart Prize, 1184
Streptomycin and dihydrostreptomycin, 1076
— in ophthalmology, 151
Surgery on television, 1344
Taste, mechanism of, 860
Television, social consequences of, 1017
Triethylene melamine in Hodgkin's disease and lymphosarcoma, 1343
Tuberculosis; a new treatment, 478
— pulmonary, and pregnancy, 862; 1030 (C)
— treatment with *iso*nicotinic acid hydrazide, 1015
Universities and money, 479 (correction, 614)
Variola minor, 478
Vitamin-A poisoning, 539
Vitamin B_{12} and growth, 1401
Water supply of London in wartime, 539
Welding and health, 1239
West Indian Medical Journal, 152
Winter epidemics, 38
Work of W.H.O., 913
Xylocaine, 211; 328 (C), 488 (C)

Anoxia and sleep levels, 449, 475 (L), 651, 971
"Antabuse." *See* Tetraethylthiuramdisulphide
"Anthisan" poisoning, treatment of, 163
"Anthisan," skin eruption due to, 1405
Anthologies: *Doctors By Themselves*, 473
— *The Quiet Art* (R. Coope), 959
Anthrax, cutaneous; treatment with chloramphenicol (P. S. Clarke), 86 (O)

ANTIBIOTICS: *See also* "Aureomycin"; Chloramphenicol; Penicillin: Streptomycin; "Terramycin": etc.
Antibiotic Therapy (H. Welch and C. N. Lewis), 1339
Dictionary of Antibiosis (L. Karel and Elizabeth S. Roach), 1290
For underweight infants, 985
Pulmonary aspergillosis following antibiotic treatment of post-influenzal bronchopneumonia (J. D. Abbott and others), 523 (O); 537 (L)
Resistance of bacteria to, 425 (L); 434; 546 (C), 712 (C)
What is an antibiotic? 1290 (L)

ANTIHISTAMINES:
"Anthisan" poisoning, treatment of, 163
— skin eruptions due to, 1405
"Antistin" in habitual abortion, 437
In non-urticarial dermatoses, and a retrial of antihistamine iontophoresis (P. Inman and I. C. Cowan), 1064 (O)
In rheumatoid arthritis, 109, 324
Mode of action and clinical use, 380

Antiseptics of quaternary ammonium type, advantages of, 985, 1260
Antisera, reactions to, 214
"Antistin" in habitual abortion, 437

ANY QUESTIONS?:
Any Questions?: First series (Book review), 260
Abortion, repeated, significance, and future outlook, 339
— therapeutic, in acute heart failure, 1260
Accommodation in ophthalmoscopy, 1042
Acne during menstruation, treatment of, 1312
Acrocyanosis, 396, 670
Agene, effect on vitamins in flour, 614
Air, purification of, 1259
Albuminuria in gout, 670
Alcohol for angina, 502
Aluminium and silicosis, 829, 1206
Amidone, 1260
p-Aminosalicylic acid, toxic effects, 58
Anaesthesia, dangerous hypotension after, 613
Analgesia for urethral instrumentation, 395
Analgesics for the nose, 285
Angina pectoris attacks precipitated by cold, 118
"Antabuse" therapy, 931
Antibiotics for underweight infants, 985
Aperients suitable for elderly patients, 502, 830
Appendicitis in infancy, 777
Appetite, perversion in infants, 778
Ascorbic acid, function in adrenal cortex, 58
Assault; mitigation, 502
Asthma, proprietaries for, 448
— treatment with oral cortisone, 1206
Autonomic ganglion block during anaesthesia, 1144
Barbiturates and blood coagulation, 1144
— first use of, 986
Bat ears, 724
B.C.G. vaccine; risks in handling, 1143
Beds in sanatoria, 1418
Bilateral renal tuberculosis, 396
Bladder control in infancy, 669
Bleeding from the umbilicus, 1092
Boy or girl?, 340
Brimstone and cancer, 448
Caesarean section, post-mortem and ante-mortem, indications for, 879, 1092
— — — legal aspects, 879
Calciferol in Boeck's sarcoidosis, 58
— in tuberculosis, 986
Camphorated oil in bronchitis, 118
Carbon dioxide slush, 230
Chemotherapy and immunity, 1260
Children: Visiting children in hospital, 613
Clinical thermometry, 1418
Cod-liver oil stains, 932
Colour vision of schoolchildren, 777
Constipated infant, 1091
Cooley's anaemia, inheritance of, 880
Coronary disease, 723
Cushing's syndrome, potassium depletion in, 1205
Cytochrome *c* in carbon monoxide poisoning, 118
Dangerous drugs in the anaesthetic room, 1365
Dangers from penetrating oils, 829
Deaf-mutism, inheritance of, 830
Death after drowning and electrocution, signs of, 1144
Diabetes mellitus: Is bread good for diabetics? 558 (correction, 830)
Diet in toxaemia of pregnancy, 723
Digitalis in toxic myocarditis, 1205
Doctor at the sink, 724
Drinking-water, storage of, 724
Eczema of hand in child, 1312, 1418
E.D.T. crystals, 1418
Emetine in scorpion stings, 396
Endocrine therapy of delirium tremens, 614
Ethyl acetate and toluene, toxic effects, 174
Eyestrain, causes and effects, 229
Fat-soluble vitamins, 1144
Fertility after prostatectomy, 670
Fits for diagnosis, 395
— African worms, 339

ANY QUESTIONS (*continued*):
Flavine rashes, 1206
Fluorescent lighting, 230
Gastric 'flu, 396
Geographical tongue, 448, 670
Glandular regression in Hodgkin's disease, 829
Gold in rheumatoid arthritis complicated by diabetes, 880
Gräfenberg ring, 1418
Hair oil, home-made, 614; 724 (C)
Hearing: Testing in schoolchildren, 117
Heartburn, 1206
Heliotherapy, 724
Hepatitis, infective, intradermal test for, 1366
Herpes gestationis, 230
Hormone therapy in endometriosis, 286
Hygiene for the medical tramp, 1365
Hypnosis and asthma, 173, 487, 546, 816
Hypnotics, quick-acting, 118, correction, 396
Infants: Adaptability to changes of climate, 286
Insemination, artificial (donor), 1259
Insulin-resistance and schizophrenia, 501
Iritis, recurrent; causes and treatment, 1366
Judo neck-strangle, 447
Keratoderma climactericum, 1206
Lactation, arrested, fate of milk after, 985
Laevulose, sobering effects of, 1041
Lead workers, surveillance of, 931
Lupus erythematosus, discoid, treatment and prognosis, 117
Marbled skin, 396
Mercurial diuretics, prolonged administration of, 931
Molluscum contagiosum, treatment, 57
Morphine, idiosyncrasy to, 502
Myelomatosis, multiple, 340, 558
Nasal sinuses, lymph draining of, 118
Neuralgia, post-herpetic, treatment, 230
Night nurses' paralysis, 557
— sweats, 932
Orgasm, absence of sensation during, 1092
Otitis media, flying and bathing, 777
Over-sexed girls, 986
Ovum, unfertilized, length of survival, 285
Paronychia, chronic, treatment, 58
Pemphigus neonatorum, notification of, 448
Penicillin: Discoloration in syringes, 1205
Perthes's disease, prognosis in, 1311
Pituitary irradiation for diabetes, 1418
Pneumonectomy, first successful, 986
— for bronchiectasis, 557
Pneumonias, apical, meningeal signs in, 829
Pregnancy tests, 501; 724 (C)
— accuracy of various tests compared, 1366
Pregnenolone in arthritis, 1206
Priapism, neonatal, 614
Proctalgia fugax, 669, 710, 880
Psycho-analysis, Adlerian and Freudian compared, 447
Psychological effects of female castration, 557
Quaternary ammonium antiseptics, 985, 1260
— — compounds, in disseminated sclerosis, 880
Rabies control, 931
Rectum, congenital absence of, treatment of, 558; 762 (C), 1084 (C)
Redheads, 1143, 1260
Resins for peptic ulcer, 829
Retina, detached, treatment of, 1041
Retinitis pigmentosa, prognosis in, 118
Rheumatism; treatment with gamma rays, 1311
Salt and boils, 174
Schizophrenia, physical characteristics associated with, 558, 932
Schools for the educationally subnormal, 286
Scleritis, 1417
Sclerosis, disseminated, 502
Scrotal retention cysts, 174
Seasickness, 57, 1042
Sebaceous cysts of the scalp, 777
Sensitivity to oak, 1092
Skin: Staining due to gold watch, 1042
Sodium-containing barbiturates in eclampsia, 1091 (correction, 1144)
Spirit, surgical, purification after use, 229
Stains from medicinal dyes, removal of, 1366
— on doctors' clothes, 173
Staphylococcal mastitis in cows and food-poisoning in man, 1144
Stellate ganglion block, 777
Sterilization during puerperium, 1312
Styes, recurrent, 1259
Suicide, attempted, 339, 490, 657, 761, 921
Sweethearts, 985
Syncope and fatal wasp stings, 1366
Teeth, carious, bacteriology of, 1418
— on edge, 1041
Television and the eyes, 558
Temperature, normal, 1259
Thrombocytopenic purpura and pregnancy, 932
Thrombosis, cerebral, anticoagulant therapy contraindicated, 58
Tobacco: Pills for prevention of smoking, 118
Trauma and thromboangiitis obliterans, 1143
Treatment of lightning pains, 1091, 1312
Tuberculosis and breast-feeding, 879
Tuberculous glands in the neck, 285, 830
Ulcers, trophic, after infantile paralysis, 1312
Urine, high ketosteroid level, significance of, 879
Urticaria, intractable, 1417
Vanishing creams, 670
Venesection, 1260
Vitamin B_{12}: Is it a tonic? 1041
— supplements in tuberculosis, 986
ANY QUESTIONS (*continued*):
Vitamins during lactation, 229, 396
Vulva, periodic oedema of, 1092
Watches: People on whom watches will not keep time, 1366
Water softeners and lead pipes, 1417
Wax baths, 1417
Whooping-cough and asthma, 1041
Yaws and repeated abortion, 830
Aperients suitable for elderly patients, 502, 830
APITZ, K. *See* RÖSSLE, R., 908
Appendicitis in infancy, clinical picture, 777
— in the West Indies (A. Leacock), 1347
— left-sided, 1357
Appendix, absence of, 1302
— gangrenous, 653
Appetite, perversion in infants: cause and treatment, 778
Applied Statistics, 1089
APPOINTMENTS: 116, 173, 284, 395, 556, 613, 723, 776, 828, 984, 1091, 1143, 1205, 1310, 1365, 1416
Arbitration award in the National Health Service, 697 (L), 749; 718, 771 (parliamentary notes); Working Party, 1235 (L), 1253
ARDEN, G. P.: Intra-articular streptomycin, 1194
— *See* AHERN, R. T., 466
ARDIS, J. A. *See* TATLOW, W. F. T., 1010
Argentinian congress of thoracic surgery, 1364
ARMED FORCES:
Appointments and retirements, 52, 497, 719, 825, 927, 1307
Awards and decorations, 112, 497, 719, 825, 927
Call-up of reservists, 775
King's Honorary Physicians, 332
Naval Medical Compassionate Fund, 825
Need for medical and dental officers to be members of defence body, 439
Prisoners of war, 112
Rejected recruits, 979
Royal Navy: Awards and decorations, 52
Armless gadgets for, 230, 778
ARMSTRONG, J.: Employment for over-sixty-fives, 1409
ARRAU, C. M. *See* CHATTERJEA, J. B., 987
Arris and Gale Lecture, 59
Arsenic in cigarettes (parliamentary note), 824
Arsenotherapy; purpura and pancytopenia as sequelae (M. G. Nelson), 300 (O); 714 (C)
Art: Exhibition of medical illustration at B.M.A. House, 1293, 1363
— *Medical and Biological Illustration*, 98 (A)
— Royal Academy's annual exhibition, 1038
"Artane," toxicity of, 382
ARTERIES: *see also* Embolism
Arteriosclerosis, cerebral; treatment with "priscol," 98 (A); 106 (C)
Occlusion, spontaneous, during epileptic fit (W. L. B. Leese), 854; 1303 (C)
Spasm, traumatic; physiology and relief (J. B. Kinmonth), 59 (O); 93 (L)
ARTHRITIS:
Arthritis and Rheumatism Foundation, U.S.A., 341
Osteoarthritis, generalized, and Heberden's nodes (J. H. Kellgren and R. Moore), 181 (O); 208 (L); 383 (C), 765 (C)
Polyarthritis, acute, with pulmonary consolidation and pleural effusion (A. W. Lees), 246 (O); (correspondence), 765
Pregnenolone in arthritis, 1206
Rheumatic diseases in the U.S.A. (J. J. R. Duthie), 341 (O)
Rheumatoid (W. S. C. Copeman), 1185
— antirheumatic effects of hydrocortisone (free alcohol), hydrocortisone acetate, and cortisone (free alcohol) as compared with cortisone acetate; results from oral administration in patients with rheumatoid arthritis (E. W. Boland), 559 (O); 588 (L)
— complicated by diabetes, gold therapy in, 880
— critique of therapy, 602
— effect of antihistaminics, 109, 324
— effect on bleeding-time of an extract of blood of patients with rheumatoid arthritis (R. Greene, Josephine Vaughan-Morgan, and Julia Gammon), 17 (O); 438 (C), 603 (C)
— observations on prolonged cortisone administration (W. S. C. Copeman and others), 397 (O); 601 (C)
— responsiveness to A.C.T.H. and cortisone (J. H. Kellgren and others), 997 (O); 1016 (A) (correction, 1144)
— treatment with wax baths, 1417
Artificial limbs, charge for (parliamentary note), 552
Ascorbic acid, function in adrenal cortex, 58
Aspergillosis, pulmonary, following antibiotic treatment of post-influenzal bronchopneumonia (J. D. Abbott and others), 523 (O); 537 (L)
ASPIN, J.: Shortage of *x*-ray films, 327
Assault: Mitigation plea in presence of pre-existing abnormality, 502
Association of British Pharmaceutical Industry; annual dinner, 982
Association of Clinical Pathologists; 48th meeting, 1039
See also
Aero Medical Association
British Association of Allergists
British Association of Otolaryngologists
British Association of Physical Medicine
ASSOCIATION (*continued*):
See also
British Epilepsy Association
British Medical Association
International Association of Gerontology
International Fertility Association
Medical Association for the Prevention of War
Scottish Western Region Association of Psychiatrists
ASTHMA:
Aetiology, 1
Attack precipitated by whooping-cough, 1041
Management of Bronchial Asthma: A Guide to Treatment (H. G. J. Herxheimer), 1394
Prescription of proprietary drugs for, 448
Somatic and Psychiatric Treatment of Asthma (Editor: H. A. Abramson), 1288
Treatment with hypnosis, 173, 326, 487, 546, 816, 922
— with oral cortisone, 1206
X-ray appearances in; 200 cases (H. Royle), 577 (O)
Athens, School of Hygiene, reorganization of, 808
ATKIN, I.: Discharge of certified mental patients, 1303
ATKINSON, E. C.; Fouling the nest, 1195
ATKINSON, M.: Value of histamine skin test in Ménière's disease, 869
ATKINSON, W. B., and others: *The Anatomy of the Gorilla*, 365
Atomic structure; basic principles (Sir James Walton), 99
"Aureomycin"; local application for aphthous stomatitis, 1082, 1246
— in tetanus neonatorum, 470
"Aurofax," response of megaloblastic anaemias of pregnancy to, 852
Australasian Annals of Medicine, 115
Austria: Rehabilitation, 555
Autografting, traumatic, 274
Auxiliaries, medical, register of, 1309
Axilla, pilonidal sinus of (I. Aird), 902 (O); 1356 (C)
AYRE, P.: T-piece anaesthetic technique, 327

B

BACH, F. *See* LING, T. M., 756
Backache, clinical assessment of (J. G. Bonnin), 540
Bacteria at the sea bottom, 1343 (A)
Bacterial resistance to chemotherapy, 425 (L); 546 (C), 712 (C)
Bacteriological warfare in Korea, 65?
Bacteriology: *Medical Bacteriology*, 5th ed. (Sir Lionel Whitby), 1072
— *Recent Advances in Bacteriology*. Edited by J. H. Dible. 3rd edition by J. D. Maclennan, 585
BADENOCH, J.: Cortisone in idiopathic steatorrhoea, 356 (O)
BAENSCH, W. E. *See* SCHINZ, H. R., 586
BAGRATUNI, L.: Cephalic tetanus, with report of case, 461 (O)
BAILEY, A. *See* CRAWFORD, O. B., 1083
BAILEY, N. L. *See* SORSBY, A., 119
BAIRD, D.: Social trends and home confinements, 1246
BAIRD, J. P. *See* MORRISON, R. J. G., 198
BAIRD, William Joseph McLearn, obituary of, 224
BAKER, C., BROCK, R. C., CAMPBELL, M., and WOOD, P.: Valvotomy for mitral stenosis; further report, on 100 cases, 1043 (O); 1073 (L)
BAL therapy in mercury nephrosis, 358
Balantidiasis in South Persia (A. G. McCarey), 629 (O); 1357 (C)
BALME, H.: Oesophagitis, 1250
"Banocide." *See* Hetrazan
BANSI, H. W.: *Thyreotoxikosen und Antithyreoidale Substanzen*, 1339
Banti, Guido, centenary of birth of, 1335
Barbiturates; effect on blood coagulation, 1144
— first use of, 986
— relation between sensitivity to thiopentone, sulphonamides, and sunlight (F. Evans and G. Gould), 417 (O); 477 (A)
— sodium-containing, in treatment of eclampsia, 1091 (correction, 1144)
Barium enema; death following use in child with Hirschsprung's disease (A. Jolleys), 692 (O); (correspondence), 866
BARLEY, D. A.: Plastic artificial gullets, 1358
BARLEY, R. G. *See* MORRIS, J. N., 503
BARLOW, F.: Physical factors affecting the efficiency of insecticides, 543
BARNARD, H. F.: Sterilization of woollen fabrics, 21 (O); 601 (C)
BARNES, J. M.: Insecticides toxic to man, 544
BARNETT, E. E. *See* GELFAND, M., 1172
BARNS, H. H. F.: Mucus extractor for use on newborn infant, 598
— and MORGANS, M. E.: Conduct of pregnancy complicated by diabetes mellitus, 1058 (O); 1248 (C)
BARON, C. F. J.: Qualifications of coroners, 921
BARRETT, A. M., GAIRDNER, D., and MCFARLAN, A. M.: An outbreak of encephalitis possibly due to poliomyelitis virus, 1317 (O)
BARSON, G. J. *See* SHREWSBURY, J. F. D., 954
BARTER, R. W.: Antihistaminics in rheumatoid arthritis, 109
BARTHELS, A. C.: Nature cure, 818
BARTHOLOMEW, A. A.: Salt intake and prickly heat, 819

BARTLETT, Sir Frederic: Fatigue, 814
Barwell, Claud Foster, appointed to Goldsmiths' Company's Chair of Bacteriology at London Hospital Medical College, 1361
Bat ears, 724
BATCHELOR, R. C. L., and MURRELL, Marjorie: *Venereal Diseases described for Nurses*, 365
BAUER, A. W.: Acrocyanosis, 670
BAYNES, T. L. S.: *Handbook of Gynaecology*, 638
B.C.G.: Mass vaccination in Malta; report on the B.C.G. vaccination campaign in 1950 (V. Zammit-Tabona), 837 (O)
— tuberculosis in families with B.C.G.-vaccinated and non-vaccinated children (A. W. Dickie), 682 (O); 1194 (C)
— vaccination, 270
— — efficacy of, 147 (L)
— vaccine; risks in handling, 1143
BEACH, F. A. *See* FORD, C. S., 1338
BECK, A.: Emergency operation on patient under A.C.T.H. and cortisone therapy, 636; 1303 (C)
Bedding, hygienic (parliamentary note), 824
BEDFORD, P. D.: Side-effects of a preparation of vitamin B_{12}, 690 (O)
BEDFORD, T.: Environmental factors in fatigue, 816
Beds. *See* Hospitals: Beds
BEDSON, S. P., awarded Conway Evans Prize, 979
Bees: Division of labour and food transmission within the honeybee community, 1389
Begley Prize, 875
Belfast University. *See* Universities: Belfast
BEMBRIDGE, B. A., COXON, M., HOULTON, A. C. L., JACKSON, C. R. S., and SMALLPEICE, V.: Retrolental fibroplasia; problem of prematurity, 675 (O); 698 (L); 924 (C), 1029 (C)
BENNETT, A. H., and others: *Phase Microscopy: Principles and Applications*, 1117
BENNETT, Bethina A.: *Guide to Professional Nursing*, 960
BENSTEAD, Nancy, and THEOBALD, G. W.: Iron and "physiological" anaemia of pregnancy, 407 (O); 476 (A); 599 (C), 817 (C)
Benzodioxane test, negative, in presence of phaeochromocytoma (H. P. Tulloh), 531 (O)
2-Benzyl-imidazoline hydrochloride. *See* "Priscol"
BERESFORD, O. D. *See* WATSON, T. A., 107
Beriberi, acute, complicating polyserositis; case in Britain (J. Thomson and K. R. Keay), 295 (O)
BERNAL, J. D.: *The Physical Basis of Life*, 32
BERNSTIEN, A. E.: Hypnotism in medicine, 816
BERRY, R. J. A.: Growth and development of children, 1133
BERRY, W. T., COWIN, P. J., and MAGEE, H. E.: Haemoglobin levels in adults and children, 410 (O)
BERTWISTLE, A. P.: When should gall-stones be removed,? 1031
— Myositis ossificans, 1357
BETT, D. H.: Proctalgia fugax, 880
BETT, W. R.: *Osler: The Man and the Legend*, 1178
BEVAN, A.: *In Place of Fear* 749 (L); (correspondence), 870, 974, 1030
— on the adjudication award (parliamentary note), 772
— and the Spens Committee, 1291 (A)
BEVIS, D. C. A.: Habitual abortion, 440, 819
BHATTACHARYA, B.: Diabetes and pregnancy, 1248
BICKFORD, J. A. R. *See* TATLOW, W. F. T., 1010
BIDSTRUP, P. L., and HUNTER, D.: Toxic chemicals in agriculture, 277
— *See* HUNTER, D., 1135
BIER, A.: *Das Leben*, 1178

BILHARZIASIS:
And death of snail vectors, 820
In the West Nile district, 158
"Nilodin" in treatment of *Schistosoma haematobium* (M. A. Haseeb), 1331 (O)
Peculiar syndrome of bladder paralysis in natives of South Central Africa (M. Gelfand and E. E. Barnett), 1172 (O); 1408 (C)
Persistent urinary *Salm. dublin* carrier with (W. S. Miller), 628 (O)
Schistosoma infection as cause of fits, 339

BILSBARROW, G.: Angina in cold weather, 558
BINGOLD, A. C. *See* CLARK, J. M. P., 903
BINNIE, M. C.: Geriatrics, its scope and problems, 650
BINNING, R.: Prolonged respiratory paralysis after succinylcholine, 971

BIOGRAPHY:
And They Shall Walk. The Autobiography of Sister Elizabeth Kenny, 802; 1082 (C)
Between Life and Death (H. Williams), 696
Born Twice (Olga Franklin), 1011
Chambers's Dictionary of Scientists (A. V. Howard), 310
Circus Doctor (J. Y. Henderson), 1118
Davidson, William, of Aberdeen by J. Read (C. Singer) (Nova et Vetera), 380
English Pioneers of Anaesthesia (*Beddoes, Davy, and Hickman*) by F. F. Cartwright, 706
Gordon, Mervyn Henry; eightieth birthday, 1350
Handley, W. Sampson, eightieth birthday of, 812
Langdon-Brown, Sir Walter (G. Evans), 175 (O); 206 (L)
Leonardo da Vinci, 804 (L); 1142
Life and Work of Astley Cooper (R. C. Brock), 1071
Physician as Man of Letters, Science, and Action (T. K. Monro), 959

BIOGRAPHY (*continued*):
Symcotts, John; a Seventeenth Century Doctor and his Patients (F. N. L. Poynter and W. J. Bishop), Bedfordshire Historical Record Society Publications, Vol. XXXI (Nova et Vetera) (C. Singer), 1351
Under the Sun. A Memoir of Dr. R. W. Burkitt, of Kenya (J. R. Gregory), 311
Dr. Viper. The Querulous Life of Philip Thicknesse (P. Gosse), 1338

Biological warfare, 446, 652
Biology: *Das Leben* (A. Bier), 1178
— *The Physical Basis of Life* (J. D. Bernal), 32
Biophysics: *Progress in Biophysics and Biophysical Chemistry*, vol. 1 and 2 (Editors: J. A. V. Butler and J. T. Randall), 856
BIRBECK, Angela. *See* WYNN, A., 893
BIRCH, C. A.: Safety adapter for syringes, 1029
BIRD, J. D. H.: Explosion during anaesthetic, 1083
BIRKS, P. H.: Megaloblastic anaemia in Indians, 764
Birmingham: *City of Birmingham Annual Abstract of Statistics. No.* 1. 1931–1949 (Editor: R. Padley), 908
— Role of statistics in regional hospital planning, with special reference to Birmingham Hospital Region (Vera Norris), 129 (O); (correspondence), 272, 437
— University. *See* Universities: Birmingham
BIRNIE, C. R.: Discharge of certified mental patients, 971

BIRTH CONTROL:
Discussion at W.H.O. Annual Assembly, 1157, 1405
Gräfenberg ring, 1418
Memorandum on family planning, with particular reference to contraception, 595 (correspondence), 708, 766, 867, 970, 1027, 1132, 1247, 1354
Oral contraceptives, 1183 (A)
Payment for advice, 597

Birthday Honours, medical, 1299; 1293 (A)
BISHOP, P. M. F.: Habitual abortion, 923
— and RICHARDS, N. A.: Habitual abortion; further observations on the prophylactic value of progesterone pellet implantation, 244 (O); 428 (L); (correspondence), 437, 440, 604, 819, 923
— *See* COPEMAN, W. S. C., 397
BISHOP, Thomas Henry, obituary of, 664
BISHOP, W. J. *See* POYNTER, F. N. L., 1350
BLACK, J. M.: Ruptured spleen, 223
Black's Medical Dictionary, 696
Bladder, control in infancy, 669
— hour-glass (A. E. Roche), 583
— paralysis; peculiar syndrome in natives of South Central Africa (M. Gelfand and E. E. Barnett), 1172 (O); 1408 (C)
— tumours in dyestuff workers, 1123 (A)
BLADES, A. N. *See* WEBSTER, D. D., 307
Bland-Sutton, Lady, bequest to Royal College of Surgeons, 875, 979, 1200, 1251
BLANEY, P. J.: Value of cigarettes after operation, 1354
Blankets, sterilization of (H. F. Barnard), 21 (O); 601 (C)
BLEULER, E.: *Dementia Praecox or the Group of Schizophrenias*, 32, 326

BLINDNESS:
Annual Report of the Guide Dogs for the Blind Association, 1069
Campaign in East Africa, 393
Curable, common causes of (J. Foster), 318; 490 (C), 651 (C), 762 (C), 1083 (C)
In Southern Nigeria (G. E. Dodds), 584; 1248 (C)
In Uganda, 159
Onchocerciasis in West Africa, 853
Significance of Heredity in Ophthalmology. A Tasmanian Survey (J. B. Hamilton), 803
Traumatic, causes and prevention, 318

BLOCH, Carl Edvard, obituary of, 663

BLOOD: *see also* Agranulocytosis; Anaemia; Leukaemia; etc.
A.C.T.H. and cortisone in treatment of blood disorders; preliminary report to Medical Research Council by panel on haematological applications of A.C.T.H. and cortisone, 1261 (O)
Anticoagulants in leukaemia, 162
Bleeding time; effect of extract of blood of patients with rheumatoid arthritis (R. Greene, Josephine Vaughan-Morgan, and Julia Gammon), 17 (O); 438 (C), 603 (C)
Circulation, effect of *nor*adrenaline and adrenaline on (H. J. C. Swan), 1003 (O)
— reciprocal changes in blood flow of the limbs and rectum during general anaesthesia (R. Shackman and I. G. Graber), 1284 (O); 1409 (C)
Coagulation: *Die Blutgerinnung in Theorie und Praxis* (J. Schmid), 92
— clotting mechanism, 1229
— effect of barbiturates on, 1144
— prolonged anticoagulant therapy with heparin (A. Wynn, J. F. Goodwin, and Angela Birbeck), 893 (O)
— reciprocal changes in blood flow of the limbs and rectum during general anaesthesia (R. Shackman and I. G. Graber), 1284 (O)
Effect of cortisone on, 402, 1261
Erythrocyte sedimentation rate; anomaly in rheumatic diseases (Sylvia Goldberg, L. E. Glynn, and E. G. L. Bywaters), 202

BLOOD (*continued*):
Erythrocyte sedimentation rate, correction in anaemia; experimental study based on interchange of cells and plasma between normal and anaemic subjects (J. C. F. Poole and G. A. C. Summers), 353 (O); 369 (A)
— — — effect of cortisone on, 402
— — — Westergren and Wintrobe methods compared, 277
Erythrocytes, effect of hypochromasia on specific gravity and sedimentation of (K. B. Rogers), 1109 (O) (correction, 1418); 1407 (C)
Fourth International Congress of Haematology, 115
Groups: *Human Blood Groups and Inheritance* (Sylvia D. Lawler and L. J. Lawler), 856
— *Human Blood Groups Utilized in Disputed Paternity Cases and Criminal Proceedings* (P. H. Andresen), 1395
— kernicterus without prematurity or blood-group incompatibility (N. R. Butler and W. G. Spector), 1168 (O)
— *Rh Blood Groups and their Clinical Effects*, 667; 870 (C)
— study on the A factor as a cause of haemolytic disease in premature babies (S. Phansomboon and O. J. Pollak), 1170 (O)
— verbal usage of the CDE notation for the Rh blood groups (S. Haberman and J. M. Hill), 851 (O)
Haemoglobin levels in adults and children (W. T. C. Berry, P. J. Cowin, and H. E. Magee), 410 (O)
Hämolytische Syndrome im Kindesalter (C. Gasser), 857
Handbook of Diseases of the Blood (A. Piney), 423
Oxygen saturation at various levels of sleep, 449
Pancytopenia and purpura complicating arsenotherapy (M. G. Nelson), 300 (O); 714 (C)
Pressure, effects of hexamethonium on, 252
— effects of "methedrine" and L-*nor*adrenaline, 106
— high. *See* Hypertension
— low, after anaesthesia, 613
— methods of reduction, 935
— L-*Nor*adrenaline as a vasoconstrictor, 546
— reduction by pre-operative arteriotomy, 965 (A)
Proceedings of the Third International Congress of the International Society of Hematology (Editor-in-Chief: C. V. Moore), 1072
Prothrombin time, effect of vitamin-K preparations on, 412
Red cells. *See* Blood, erythrocytes
Sickle-cell trait in Southern India (H. Lehmann and Marie Cutbush), 404 (O); 426 (L); (correspondence), 920
— — in Western Nigeria: survey of 1,881 cases in the Yoruba (D. B. Jelliffe and J. Humphreys), 405 (O); 426 (L); (correspondence), 763, 920
Stains, removal of, 173
Transfusion; amount of blood consumed by banks, 136
— *Blood Transfusion in Clinical Medicine* (P. L. Mollison), 472
— conference, 500
— — international, on standardization and interchangeability of equipment, 624
— death from toxaemia after (medico-legal note), 1036
— exchange transfusion in severe anaemia (T. Ward), 631 (O)
— Oliver Memorial Fund fifth award, 1364
— use of plasmapheresis in donors (J. A. Grifols-Lucas), 854
Vessels, cerebral; treatment of embolism and thrombosis (R. Leriche), 231 (O); 262 (L); 381 (C), 486 (C), 487 (C); 777
— physiology and relief of traumatic arterial spasm (J. B. Kinmonth), 59 (O); 93 (L)

BLOOM, P. M.: Sexual disorders in women, 656
BOASE, A. J.: Blindness in Uganda, 159
BOCOCK, E. J., and HAINES, R. W.: *Applied Anatomy for Nurses*, 424
BODENHAM, D. C.: Metric system in Britain, 819
BODIAN, M., WHITE, L. L. R., CARTER, C. O., and LOUW, J. H.: Congenital duodenal obstruction and mongolism, 77 (O); (correspondence), 276, 384
Boils, recurrent, and excessive salt intake, 174
BOLAND, E. W.: Antirheumatic effects of hydrocortisone (free alcohol), hydrocortisone acetate, and cortisone (free alcohol) as compared with cortisone acetate; results from oral administration in patients with rheumatoid arthritis, 559 (O); 588 (L)
BOLD, W. J.: Music while you operate, 273

BONE:
Acrylic replacement after segmental resection (J. M. P. Clark and A. C. Bingold), 903 (O)
Fractures in the aged (R. H. Metcalfe), 1240
Hyperostosis, cortical, infantile (W. J. Matheson and Mary Markham), 742 (O)
— Marrow, effect of repeated injections of A.C.T.H. on (G. Hudson, G. Herdan, and J. M. Yoffey), 999 (O); 1135 (C)
— — sclerosis with polycythaemia, 1113
Ossification, accelerated, in cretin caused by high thyroid dosage (T. Colver and T. Lodge), 89

BONNAR, A.: Post-mortem and ante-mortem caesarean section, 1092
BONNIN, J. G.: Clinical assessment of backache, 540
Books received, 33, 92, 146, 205, 261

BORG, C.: Tragedy of involutional depression, 1303
Bornholm disease; orchitis as complication (R. J. G. Morrison and J. P. Baird), 198 (O)
— — outbreak in West Fife practice (W. W. W. McNeish and C. Stewart), 744 (O)
BOUCHER, P. R.: Sexual disorders in women, 273
BOUND, J. P. *See* FINLAY, H. V. L., 1134
BOURNE, A.: Ovarian dysmenorrhoea, 272
BOURNE, G.: Mitral valve disease in the light of surgical treatment, 896 (O)
— Heart block, 966
BOURNE, G. H. (Editor): *Cytology and Cell Physiology*, 2nd ed., 747
— Copper content of foods, 1193
BOURNE, I. H. J.: Wrist splint, modified, 383
BOURNE, W. A.: Nomenclature of diseases and operations, 330
BOUSFIELD, G.: Educating the public about cancer, 1409
BOWER, W. D.: Preventive medicine, 327
BOWERS, R.: Skin rash at Cheltenham, 1303
BOWMAN, T.: Tale of three sneezes, 1358
BOYDE, Harry Caine, obituary of, 926
BOYD-ORR, Lord, presentation to, 1257
BRAASCH, W. F., and EMMETT, J. L.: *Clinical Urography: An Atlas and Textbook of Roentgenologic Diagnosis*, 1011
BRACHFELD, O.: *Inferiority Feelings in the Individual and the Group*, 585
Brachial plexus paralysis, post-operative (F. G. Wood-Smith), 1115
Braille, Louis, centenary of death of, 56
BRAILSFORD, J. F.: Shortage of *x*-ray films, 223
BRAIN, R. T.: Herpes zoster and varicella, 651
BRAIN, W. R.: *Mind, Perception, and Science*, 856
— Committee on poliomyelitis, 1131
— knighthood conferred on, 44
— re-elected President, Royal College of Physicians of London, 862

BRAIN:
Arteriosclerosis treated with "priscol," 98 (A); 106 (C)
Brain as a machine (Sir Henry Cohen), 542
Brain Metabolism and Cerebral Disorders (H. E. Himwich), 802
Embolism and thrombosis of cerebral vessels; treatment (R. Leriche), 231 (O); 262 (L); 381 (C), 486 (C), 487 (C); 777
Injuries; Kroll's treatment (parliamentary note), 609
Intracranial haemorrhage, spontaneous, in the young, 1013
Leucotomy. *See* Leucotomy
Meningeal signs in apical pneumonias, 829
Tumours; homolateral pyramidal signs with rumours of posterior fossa (L. C. Oliver and J. R. Gibbs), 634 (O)
— location with isotopes, 428 (A); 549 (C)

BRAITHWAITE, Eldred Curwen, obituary of, 389, 718
BRAITHWAITE, J. V.: Damnable heresy?, 973
BRAMWELL, Edwin, obituary of, 768, 821
BRAMWELL, J. C.: *Approach to Cardiology*, 907
BRAND, Elizabeth N.: Bacteriology of vaginal flora after use of internal tampons, 24 (O)
BRAUER, E.: Treatment of hypertension, 1191
BRAUER, Ludolph, obituary of, 51
Bread crust, fatal (medico-legal note), 1361
Breast-feeding and tuberculosis, 879
Breast-milk, vitamin content of, 229, 396
BREND, G.: *My Dear Holmes. A Study in Sherlock*, 146
BRENNAN, S.: Incontinence in a woman, 110
BREWERTON, D. A. *See* LUTWYCHE, V. U., 1302
BRIGGS, D.: Future use of cortisone and A.C.T.H. in general practice, 103 (correction, 230)
Bristol University. *See* Universities: Bristol
British Association of Otolaryngologists, annual general meeting, 227
British Association of Physical Medicine, 56
British Columbia University. *See* Universities: British Columbia
British Epilepsy Association, 324

BRITISH MEDICAL ASSOCIATION:
Annual meeting, Dublin; scientific exhibition, 371 (A), 379, 605
Film catalogue, 152 (A)
H.M. Queen Elizabeth as Patron, 1340 (L)
Law in Relation to the Illegitimate Child, 271; 265 (A)
Loyal Address to the Queen, 482
Stewart Prize awarded to C. H. Andrewes, 1184 (A)
Thomas, J. W. Tudor, as President 1953–4, 1181 (A)

BRITTON, C. J. C.: Clinical use of antihistaminic drugs, 381
Broadmoor; costs of maintenance of patients (parliamentary note), 1139
— inquiry (parliamentary note), 1086
BROCK, R. C.: *Life and Work of Astley Cooper*, 1071
— *Lung Abscess*, 1287
— *See* BAKER, C., 1043
BRODRIBB, H. S., MCMURRAY, J., and SCOTT, L. G.: Two cases of diabetes mellitus in infants under 1 year, 1060 (O); 1354 (C)
BROMAGE, P. R.: L-*Nor*adrenaline as a vasoconstrictor, 106
— Xylocaine, 328
Bronchiectasis; indications for and against pneumonectomy, 557
— putrid, in unsuspected foreign body (J. S. Glennie, J. F. Dark, and D. C. Lindars), 1009
Bronchus, foreign body as unsuspected cause of bronchiectasis (J. S. Glennie, J. F. Dark, and D. C. Lindars), 1009
BRONOWSKI, J.: *Common Sense of Science*, 653
Broughton, A. D. D., on prices in advertisements of drugs (parliamentary note), 1361
BROWN, A., and DOUGLAS, A. S.: Treatment of hypoprothrombinaemic haemorrhage, 763
— *See* DOUGLAS, A. S., 412
BROWN, A. I. P. *See* LING, T. M., 756
BROWN, C. E.: Unsuspected gangrenous appendix, 653
BROWN, C. P.: Cost of anaesthetic techniques, 546
BROWN, R. J. K.: Ferrous sulphate poisoning in children, 348
BROWN, Samuel Stuart, obituary of, 496
BROWN, William: Hypnotism in medicine, 487
— obituary of, 1136
BROWNE, J. C. McC.: Trial labour, 1025
BROWNE, R. C.: Fatigue, 814
BROWNLIE, Andrew, obituary of, 111
BRYCE-SMITH, R.: Laryngoscope blade for infants, 217
BUCHAN, J. F.: Adrenal cortical function at death, 1249
Buckston Browne Prize Essay, 55
BUDDEN, F. H.: Blindness in Northern Nigeria, 1248
BULL, G. M., appointed Professor of Medicine, Queen's University, Belfast, 1307
BULL, J. P., and JACKSON, D. M.: Treatment of burns—I, 1018; II, 1078
BULLOUGH, Fred, obituary of, 823
BULLOUGH, J.: T-piece technique for general inhalational anaesthesia; safe method of reducing operative bleeding, 28 (O); 327 (C), 384 (C), 546 (C), 654 (C)
BULMAN, J. F. H.: Acute phlegmonous colitis, 309
BURDON, J. F.: Explosion during anaesthesia, 972
BURGE, H.: Hydrostatic bag for dilatation of the cardia in achalasia, 1245
BURKE, J.: Simultaneous haemorrhage and perforation of Meckel's diverticulum, 221
BURKITT, R. T.: Post hoc, propter hoc reasoning, 221
BURKITT, R. W., biography of (J. R. Gregory), 311
BURN, J. H.: Enzyme at sympathetic nerve endings, 784 (O); 861 (A)
BURNETT, C. W. F.: Treatment of septic abortion, 886 (O); 911 (A)
BURNS, T. H. S.: Syringe service for a department of anaesthetics, 1127
Burns, prevention of (parliamentary note), 665
— treatment of (J. P. Bull and D. M. Jackson), 1018, 1078
BURROWS, H. J., and COLTART, W. D.: *Treatment by Manipulation*, 2nd ed. fully revised, 1233
BURSTON, S. R., appointed K.B.E., 44
Bus drivers, selection of, 150 (A)
BUSVINE, J. R.: Newer insecticides, 543
BUTLER, J. A. V., and RANDALL, J. T. (Editors): *Progress in Biophysics and Biophysical Chemistry*, vol. 1 and 2, 856
BUTLER, N. R., and SPECTOR, W. G.: Kernicterus without prematurity or blood-group incompatibility, 1168 (O)
BUXTON, P. A.: Insecticides today, 544
B.W. 50–63. *See* "Daraprim"
BYWATERS, E. G. L. *See* GOLDBERG, Sylvia, 202

C

Caernarvonshire and Anglesey Paediatric Club, 555
Caesarean section, post-mortem and ante-mortem indications for, 879, 1092
— — post-mortem, legal aspects of, 879
Calciferol therapy in Boeck's sarcoidosis, 58
— with streptomycin and P.A.S. in tuberculosis therapy, 986
CALNAN, W. L.: Incontinentia pigmenti, 277
CALWELL, H. G.: Debauchery of honest words, 548
Cambridge University. *See* Universities: Cambridge
CAMERON, D. M.: Hyperpiesis and hyperpiesia, 870
CAMERON, I. G.: Quinine in malaria, 1134
CAMERON, J. D.: Dermatitis after using "pyrotenex," 1249
CAMERON, K. *See* LEYS, D., 191
Camoquin therapy in malaria, 569, 572
CAMPBELL, A. J. M., GRAHAM, J. G., and MAXWELL, R. D. H.: Treatment of hypertension by oral methonium compounds, 251 (O)
CAMPBELL, A. M. G.: Medical aspects of road safety, 489
CAMPBELL, H., JOSEPHS, S., and SNAITH, L.: Growth of the foetal head, 1408
CAMPBELL, Maurice. *See* BAKER, C., 1043
CAMPBELL, Meredith: *Clinical Pediatric Urology*, 1071
Camphorated oil in bronchitis, 118
Canada: Catalogues of micro-organisms, 338

CANCER:
Adrenal cortex, carcinoma of, with endocrine manifestations; case (R. J. Harrison and D. Abelson), 303 (O); (correspondence), 655
Educating the public, 107, 276, 381, 544, 656, 824 (parliamentary note), 1301, 1409
Foot, fibrosarcoma of, 958
Hypopharyngeal cancer and its surgical treatment (R. W. Raven), 951 (O); 961 (L); 1358 (C)
Imperial Cancer Research Fund jubilee, 968

CANCER (*continued*):
Infection of secondary carcinoma by *Salm. montevideo* (F. O. Graham and P. N. Coleman), 1116
Oesophagus; diagnosis and surgical treatment of 159 cases (R. H. Franklin and J. J. Shipman), 947 (O); 961 (L)
Pensions for disablement or death of Armed Forces personnel, 391
Progress of cancer, 1119 (L); 1304 (C)
Research, funds for (parliamentary note), 773
Royal Cancer Hospital; change of name suggested (parliamentary note), 1360
Spleen, sarcoma of (P. Story and W. E. Gibb), 990 (O)
Stomach, carcinoma of (B. F. Swynnerton and S. C. Truelove), 287 (O); 313 (L); 492 (C), 708 (C), 1029 (C), 1301 (C)
— — and acute perforation, 164, 330
— simple gastric ulcer and carcinoma, 221
Sulphur taste and smell as diagnostic sign, 448
Treatment, efficiency of, 1029, 1301
— secret remedy (Rees Evans "cure"), 824 (parliamentary note); report of investigating committee, 1236 (L); 1253 (parliamentary note)
— with bacterial products, 429 (A)

Cannulae, plastic, introducer for (J. V. Mitchell) (preparations and appliances), 435
Capillary resistance and adrenocortical activity; further observations (H. N. Robson and J. J. R. Duthie), 994 (O)
Carbon dioxide acetone slush, preparation of, 230
Carbon Dioxide Therapy. A Neurophysiological Treatment of Nervous Disorders (L. J. Meduna), 1072
Carbon monoxide poisoning, cytochrome *c* as antidote, 118
CARCINOMA. *See* Cancer; and under organs and regions
CARDEN, William Alfred, obituary of, 111
Cardia, achalasia of, effect of barium meal in, 438
— hydrostatic bag for dilatation of (H. Burge), 1245
Cardiology. *See* Heart
Carditis, rheumatic, treated with cortisone; some preliminary observations (E. N. Chamberlain, J. D. Hay, and D. M. Freeman), 1145 (O); 1179 (L)
Caries, dental; bacteriology of, 1418
— — incidence in London school-children, 8
CARO, D. B.: Unusual case of acute pancreatitis, 1070
CARPENTER, K. Patricia, and SANDIFORD, B. R.: Epidemiology of human case of bacillary dysentery due to infection by *Shigella flexneri*, 103 Z, 142 (O)
CARR, J.: Allergy and rheumatoid arthritis, 324
— Chloramphenicol in Reiter's disease, 1358
Cars. *See* Motor-cars
CARTER, C. O.: Family planning, 867
— *See* BODIAN, M., 77
CARTWRIGHT, F. F.: *English Pioneers of Anaesthesia (Beddoes, Davy, and Hickman)* (C. Singer) (Nova et Vetera), 706
CASSELS, K.: Bequests to nationalized hospitals, 1251
Castration, female, psychological effects of, 557
CASTRO, J. DE: *Geography of Hunger*, 1234
Cataract, acquired, 320
— congenital, 319
— intra-ocular acrylic lenses for, 1121 (A)
— operations, general anaesthesia for, 1083
— *Trübungsforme der menschlichen Linse* (H. Sautter), 908
CATCHESIDE, D. G.: *Genetics of Micro-Organisms*, 907
Catha edulis for treatment of impaired memory, 222
CAUGHEY, J. E.: Radiological changes in skull in dystrophia myotonica, 137 (O)
— JAMES, A., and MACLEOD, E. K.: Hypopituitarism with pituitary tumours; cortisone in treatment of stupor associated with hypopituitarism, 1216 (O)
CAUSEY, G., appointed Sir William Collins Professor of Anatomy, 168
CECIL, R. L., and LOEB, R. F. (Editors): *Textbook of Medicine*, 8th ed., 748
Celastrus panniculata, clinical trials of, 222
Cell. *See* Cytology
"Celocurine." *See* Succinylcholine iodide
Census: Changes of population in 1951 Census (vital statistics), 226
Central Health Services Council. Report on Co-operation between Hospital, Local Authority, and General Practitioner Services, 912
Central Midwives Board, 983
Cerebrospinal fever. *See* Meningitis, cerebrospinal
Certification, medical, of cause of death, 641 (L)
Cervix uteri. *See* Uterus, cervix
CHAKRAVARTY, N. K. *See* CHAUDHURI, R. N., 568
CHALMERS, J. A.: Intra-partum uterine rupture, 604
— Torsion of Fallopian tube during pregnancy, 1251
CHAMBERLAIN, E. N., HAY, J. D., and FREEMAN, D. M.: Cortisone in rheumatic carditis: Some preliminary observations, 1145 (O); 1179 (L)
CHAMBERS, J. S. W. *See* ELLISON, N. P., 694
Chambers's Dictionary of Scientists (A. V. Howard), 310
CHAPMAN, T. L., and SUTHERLAND, J. W.: Late results of perurethral prostatic resection, 72 (O)
CHATTERJEA, J. B., ARRAU, C. M., and DAMESHEK, W.: Splenic puncture, 987 (O); 1016 (A)
CHATTERJEE, C. C.: *Human Physiology*, 857

CHAUDHURI, R. N., CHAKRAVARTY, N. K., and CHAUDHURI, M. N. R.: Chemotherapy and chemoprophylaxis of malaria; clinical trials in 500 cases and mass prophylaxis in a hyper-endemic area, 568 (O); 590 (A)
CHAUDHURI, M. N. R. *See* CHAUDHURI, R. N., 568
Chelsea Clinical Society, 55, 1142
Cheltenham; puzzling dermatological affection (vital statistics), 444, 1203; 1303 (C)
Chemistry: *The Essays of Jean Rey. A facsimile reprint of the original edition of* 1610, *with an introduction by Douglas McKie* (C. Singer) (Nova et Vetera), 1188
— *Organic Chemistry* (J. P. Wibaut), 1234
Chemotherapy and immunity, 1260
— resistance of bacteria to, 425 (L); 434; 546 (C), 712 (C)
Cheselden, W., bicentenary of death of, 827
CHESNEY, W. M.: Cough fracture in late pregnancy, 274
Chest: Funnel chest, 370 (A); 715 (C)
— *Thoracic Surgery* (R. H. Sweet), 424
Chicken-pox, confluent, with haemorrhagic symptoms; case (J. P. Marsden and W. J. Coughlan), 1066 (O)
— herpes zoster and (D.I. McCallum), 520 (O); 651 (C)
— second attacks, 710, 1030, 1358
Chilblains and hypothyroidism, 711

CHILDREN: *See also* Infants
Adoption; Jocasta complex in adoptive mothers, 817
Beds for mentally deficient children (parliamentary note), 1413
Caernarvonshire and Anglesey Paediatric Club, 555
Child Welfare Conference, 1364
Childhood factors in duodenal ulcer, 109
Children without a family; colloquium, 1415
Clinical Pediatric Urology (M. Campbell), 1071
Colour-vision testing, 777
Cruelty to (parliamentary note), 1087
Deafness; further observations on diagnosis in young children, with particular reference to the making of recommendations for special educational treatment (M. R. Dix and C. S. Hallpike), 235 (O); 264 (L)
Dental disease among 14-year-old London schoolchildren in 1947 and 1950 (Lady May Mellanby and Helen Mellanby), 7 (O)
Development of African children, 159
Effect of liver supplement on growth (J. Yudkin), 1388 (O); 1401 (A)
Growth and development, 963 (A); (correspondence), 1133
Haemoglobin levels, 410
Hämolytische Syndrome im Kindesalter (C. Gasser), 857
Handicapped, Scottish working party on education of, 1109
Healthier children (parliamentary note), 334
Hearing tests, 117
Homes for incurable children, 161, 277
Illegitimacy: *Law in Relation to the Illegitimate Child*, 271; 265 (A)
Infections, effect of "terramycin" in (B. Wolman and A. Holzel), 419 (O); 546 (C), 712 (C), 715 (C), 762 (C)
Lymphocytosis, infectious, acute; report on cases in day nursery (H. G. Dunn), 78 (O); 96 (A); 324 (C)
Management; discussion at Royal Society of Medicine, 1189
Mental defectives; lack of institutional accommodation, 277
— — schools for, 286
Night pain with fractured clavicle, 163, 273, 1408
Plantar warts, 712
School medical service, 210 (A); 490 (C)
Tuberculosis; survey in school-children in the Calder Valley (S. Keidan and others), 1390 (O); 1397 (L)
Ulster Paediatric Society, 612, 1309
Urology of Childhood (T. T. Higgins, D. I. Williams, and D. F. E. Nash), 695
Visiting children in hospital (A. Moncrieff and A. M. Walton), 43 (O); 824 (parliamentary note); for and against, 613
Visiting hours for parents of children in hospital, 1286

Chiropodists; 1951 *Register*, 31
CHISHOLM, Brock, world tour, 500
Cholecystitis, acute, 1127
— chronic, 1125
Cholesterol level in relation to chilblains, 711
Chloramine B tablets for water sterilization, 532
Chloramphenicol, pulmonary aspergillosis following administration of, 523
— treatment of cutaneous anthrax (P. S. Clarke), 86 (O)
— — of granulomatous ulcer of nose (I. B. Sneddon and J. Colquhoun), 298 (O); 491 (C)
— — of pink disease, 383, 605, 709
— — of Reiter's disease, 1358
Chloride, estimation in urine, 275
"Chloromycetin." *See* Chloramphenicol
Chloroquine therapy in malaria, 569, 571
Cholera vaccination, 271
Cholesterol: Changes in basal metabolism, serum-protein-bound iodine, and cholesterol during treatment of hypothyroidism with oral thyroid and L-thyroxine sodium (J. D. Robertson and H. F. W. Kirkpatrick), 624 (O)
— tolerance in coronary thrombosis (I. Wang), 1278 (O)
Cholinesterase, 248
— activity in human blood, determination of (W. N. Aldridge and D. R. Davies), 945 (O); 1015 (L); (correspondence), 1135
— at sympathetic nerve endings, 784
— inhibition of, 376
CHOLMELEY, J. A.: Streptomycin in the tuberculous knee-joint, 974
Chorion epithelioma, hormone levels in, 623
Christian science and health education in U.S.A., 140
CHRYSANTHIS, K.: Polycentron, 440
CHURCH, G. E.: Shortage of *x*-ray films, 600
Ciba Foundation Report for 1951, 592 (A)
CIBRIE, P., promoted Commandeur de la Légion d'Honneur, 929
Cigarettes. *See* Tobacco
Circulation. *See* Blood
CLAREMONT, C. A.: In memory of Dr. Montessori, 1304
CLARK, F. LE G., and PIRIE, N. W. (Editors): *Four Thousand Million Mouths*, 856
CLARK, H.: Laryngeal vertigo, 1250
CLARK, J. M. P., and BINGOLD, A. C.: Acrylic replacement after segmental resection of bone, 903 (O)
CLARKE, Joan S.: *Disabled Citizens*, 1071
CLARKE, P. S.: Chloramphenicol treatment o cutaneous anthrax, 86 (O)
Clavicle, fracture, with pain only in bed, 163, 273, 1408
CLAYTON, S., and ORAM, S. (Editors): *Medical Disorders During Pregnancy*, 1010
CLEMENTS, E. M. B., and PICKETT, K. G.: Height of Scotsmen, 1300
CLIFFORD, John Hudson, obituary of, 1252
Climate; adaptability of infants to changes, 286
COATES, J. C.: Prostatic obstruction and hernia, 605
COBB, C. C.: Bacterial endocarditis caused by coagulase-negative *Staph. albus*, 90
COCKBURN, W. C.: Spread of infection in the family, 968
Cod-liver oil for tuberculous patients (parliamentary note), 1254
— — stains, removal of, 932
Coeliac disease: *Management of Celiac Disease* (S. V. Haas and M. P. Haas), 32
COHEN, B. E., HALL, E. G., IRELAND, J. T., and KEIDAN, S. E.: Fantus estimation of urinary chloride, 48
COHEN, Sir Henry: Brain as a machine, 542
— Romance of words; an excursion into medical semantics, 813
Cold, common, effect of penicillin in, 385
— — familial spread of, 969
— — futility of research on, 222
— — vaccination against, 270
COLDREY, E.: Stomach tube in trachea, 382
COLE, J.: Progress of cancer, 1304
Colectomy, subtotal, in ulcerative colitis, 884
— total, in ulcerative colitis, 884
COLEMAN, P. N. *See* GRAHAM, F. O., 1116
Colic, abdominal, "epidemic," associated with steatorrhoea (M. E. M. Thomas), 691 (O)
Colitis, phlegmonous, acute (J. F. H. Bulman), 309
— ulcerative, chronic, surgical treatment of (W. B. Gabriel), 881 (O); 910 (L); 1194 (C)
— — relation to malignant change, 882
— — skin lesions in, 1407
Collagen diseases, pleuropneumonia-like organisms in, 346
College of General Practice, 1410

COLLEGES:
See Royal College of Obstetricians and Gynaecologists
Royal College of Physicians of Edinburgh
Royal College of Physicians of Ireland
Royal College of Physicians of London
Royal College of Surgeons of Edinburgh
Royal College of Surgeons of England
Royal College of Surgeons in Ireland
Royal Faculty of Physicians and Surgeons of Glasgow

Colliery medical services (parliamentary note), 978
COLLIHOLE, D. J.: Journals for Yugoslavia, 1030
COLLISON, L. W., awarded M.B.E., 115
COLON: *see also* Colectomy; Colitis
The Human Colon: An Experimental Study based on a Direct Observation of four Fistulous Subjects (W. J. Grace, S. Wolf, and H. G. Wolff), 959
Rupture, pneumatic (J. C. Comline), 745
Colonial Service careers, 393
COLONNA, P. C.: *Regional Orthopedic Surgery*, 146
Colour-vision testing of schoolchildren, 777
COLQUHOUN, J. *See* SNEDDON, I. B., 298
COLTART, W. D. *See* BURROWS, H. J., 1233
COLVER, T., and LODGE, T.: Accelerated ossification in cretin caused by high thyroid dosage, 89
COMLINE, J. C.: Pneumatic rupture of the colon, 745
Commonwealth Fund Fellowships, 929
Congenital defects in mongols, 276
— — supernumerary nipples, partial arachnodactyly, Fallot's tetralogy with right-sided aortic arch, horseshoe kidney, and fusion of two ribs; case report (I. Kessel), 470; 762 (C), 1249 (C)

CONGRESSES:
Blood: Fourth international congress, 115
— *Proceedings of the Third International Congress of the International Society of Hematology* (Editor-in-Chief: C. V. Moore), 1072
Cardiology: First European congress, 115
— Fourth Interamerican congress, 338
Council for Co-ordination of International Congresses of Medical Sciences, 283
Diabetes: International Diabetes Federation, 115, 284
International History of Medicine Society: 13th congress, 878
International Hospital Congress, 1187
Ophthalmological Society of the United Kingdom; 72nd annual congress, 1112
Radiology: International congress, 828
Royal Sanitary Institute Health Congress, 1021; 1131 (C), 1135 (C), 1196 (C), 1246 (C)
Surgery: Eighth international congress, 612
Conjunctivitis, effect of "terramycin" in, 420
— treated with streptomycin, 120
Connaught Military Hospital; conditions and accommodation (parliamentary note), 497
Constipation in infancy, 1091
Consultants: Behaviour towards hospital patients, 1195, 1244
Contraception. *See* Birth control
CONWAY, H.: Megaloblastic anaemia in young adults; aetiological importance of occult idiopathic steatorrhoea, 1098 (O)
Conway Evans prizes, 335
CONWAY-HUGHES, J. H. L.: Diagnostic unit for practitioners, 218
COOK, A. S.: Pink disease and antibiotics, 383
COOK, R. C.: *Human Fertility: The Modern Dilemma*, 145
Cooley's anaemia, inheritance of, 880
Cooper, Astley: *Life and Work of Astley Cooper* (R. C. Brock), 1071
COOPER, K. E., KERSLAKE, D. MCK., and MOTTRAM, R. F.: Vascular changes during anaesthesia, 1409
COORAY, G. H.: Spontaneous rupture of a leukaemic spleen, 693
COOPE, R.: *The Quiet Art*, 959
COPE, V. Z.: *History of the General Hospital near Nottingham* by F. H. Jacob (Nova et Vetera), 272
COPEMAN, W. S. C.: Future use of cortisone and A.C.T.H. in general practice, 103
— Rheumatoid arthritis, 1185
— and others: Observations on prolonged cortisone administration in rheumatoid arthritis, 397 (O); 601 (C)
Copeman Medal, University of Cambridge, 497
COPPARD, W. M.: Experiment in peacemaking, 818
Copper content of foods, 1193
CORKERY, R. A. P.: Frequency, 549
Cornea; *Ps. pyocyanea* infection treated with dihydrostreptomycin, 120
— ulcer, infected, treated with streptomycin, 121
Corneal grafting: Bill to make provision for use of eyes of diseased persons (parliamentary notes), 1138, 1200, 1252, 1360
— — supply of donor eyes, 544, 651
CORNER, G. W., appointed George Eastman visiting professor, University of Oxford, 111
Coronary heart disease: Aetiological relationship of prolonged hyperpiesia, 723
— — — in medical practitioners (J. N. Morris, J. A. Heady, and R. G. Barley), 503 (O); 535 (L); (correspondence), 651
Coroners, qualifications, 921

CORTISONE:
A.C.T.H. and cortisone in treatment of blood disorders; preliminary report to Medical Research Council by panel on haematological applications of A.C.T.H. and cortisone, 1261 (O)
Antirheumatc effects of hydrocortisone (free alcohol), hydrocortisone acetate, and cortisone (free alcohol) as compared with cortisone acetate; results from oral administration in patients with rheumatoid arthritis (E. W. Boland), 559 (O); 588 (L)
Asthma treated with oral cortisone, 1206
Effect on allergic diseases, 1208
Effects on capillary resistance, 995
Emergency operation on patient under A.C.T.H. and cortisone therapy (A. Beck), 636, 1303
In ankylosing spondylitis (F. D. Hart), 188 (O)
In general practice, 102
In idiopathic steatorrhoea (J. Badenoch), 356 (O)
In infective hepatitis, 860 (L)
In leprosy, 601, 602
In rheumatic carditis: Some preliminary observations (E. N. Chamberlain, J. D. Hay, and D. M. Freeman), 1145 (O); 1179 (L)
In rheumatic diseases in U.S.A., 342
In rheumatoid arthritis; long-term use, 342
— — observations on prolonged administration (W. S. C. Copeman and others), 397 (O); 601 (C)
— — response to (J. H. Kellgren and others), 997 (O); 1016 (A) (correction, 1144)
In stupor associated with hypopituitarism, 1216
Supplies, 335
Untoward Reactions of Cortisone and A.C.T.H. (V. J. Derbes and T. E. Weiss), 1072
COTTERELL, Gwyneth M.: Family advice, 1302
Cough fracture, 220
COUGHLAN, W. J. *See* MARSDEN, J. P., 1066

COULLIE, Alexander Glover, obituary of, 925
COUSINS, Christina M.: Sterilization of woollen fabrics, 601
COWAN, A.: Xylocaine, 488
COWAN, I. C. *See* INMAN, P., 1064
COWIN, P. J. *See* BERRY, W. T., 410
Cowpox; cases, 1249
COXON, M. *See* BEMBRIDGE, B. A., 675
— *See* SMALLPEICE, V., 385
CRAIG, M.: *Dublin* 1660–1860. *A Social and Architectural History*, 1395
CRAIG, W. S.: Care of the neonate; Instruction of the medical undergraduate, 1062 (O)
CRAWFORD, O. B., and BAILEY, A.: Xylocaine, 1083 (correction, 1135)
CRELLIN, Douglas, obituary of, 1252
Cremation Bill (parliamentary notes), 1253, 1360
— certificates, fees for (parliamentary note), 334
Crematoria; building authorizations, 391
— in England and Wales, 335
Cretin, accelerated ossification in, caused by high thyroid dosage, 89
CRICHTON, D.: Growth of the fœtal head, 922
— Accuracy of *x*-ray cephalometry *in utero*, 1026
CRICHTON-MILLER, H.: Attempted suicide, 921
Cricket Match, lawyers v. doctors, 1251
Criminal responsibility, 45, 50 (C)
Cripples: *Disabled Citizens* (Joan S. Clarke), 1071
— *Summary of Legislation and Directory of Organizations for the Care of the Physically Handicapped*, 195
CRISP, E. J.: Conservative treatment of disk syndrome, 217
CROFT, Phyllis G.: Goat's milk and infantile eczema, 709
CROFTON, J.: Shortage of *x*-ray films, 274
Crook Report on Opticians, 107
CROOT, H. J.: Extensive resection of the small intestine; short review, with two new cases, 195 (O); (correspondence), 654
Cross-infection; sterilization of hospital blankets (H. F. Barnard), 21 (O); 601 (C)
CROWDEN, G. P.: Fatigue, 814
CROWE, H. W.: Treatment of hypertension, 1191
CRUICKSHANK, R.: Pros and cons of immunization, 212, 268; (correspondence), 385, 714
CUNNINGHAM, J. F.: *Textbook of Obstetrics*, 534
Currency for medical treatment abroad, 1081
Cushing's syndrome, potassium depletion in, 1205
CUTBILL, L. J. *See* EDWARDS, P. W., 1224
CUTBUSH, Marie. *See* LEHMANN, H., 404
CUTHBERTSON, W. F. J.: Vitamin B_{12} and other growth stimulants, 161
CUTLER, Frederick John, obituary of, 1086
CUTLER, R.: Penicillin dangerous? 924
CUTTING, W. C. (editor): *Annual Review of Medicine*. Vol. 2, 205
CYRIAX, J.: Referred backache, 655
Cysticercosis as cause of fits, 339
Cystoscopy in the male, analgesia for, 395
Cytochrome *c* in carbon monoxide poisoning, 118
Cytology: *Cytology and Cell Physiology*, 2nd ed. (Editor: G. H. Bourne), 747
— *Elements of Bacterial Cytology*, 2nd ed. (G. Knaysi), 747

D

Dacryocystitis, acute, treated with streptomycin, 121
DAKIN, Henry Drysdale, obituary of, 441
DALLEY, G.: Seasickness, 448
Damages for personal injuries (medico-legal note), 605
DAMANT, H. G.: Incubation of chicken-pox, 1358
DAMESHEK, W. *See* CHATTERJEA, J. B., 987
DANAHER, J. G.: Ethics of leucotomy, 1028
Danckwerts award: Preparing the case for adjudication, 263 (L); adjudication, 697 (L), 749; (parliamentary notes), 718, 771; Working Party, 1235 (L); distribution, 1253
DANCY, J.: Left hand, right hand, 763
DANIELS, M., and HILL, A. B.: Chemotherapy of pulmonary tuberculosis in young adults; analysis of the combined results of three Medical Research Council trials, 1162 (O), 1179 (L)
"Daraprim" (B.W. 50–63); new antimalarial; trials in human volunteers (L. G. Goodwin), 732 (O), (correction, 870), 1075 (A)
— in treatment of malaria; study of its effects in falciparum and quartan infections in West Africa (I. A. McGregor and D. A. Smith), 730 (O)
DARK, J. F. *See* GLENNIE, J. S., 1009
David Anderson-Berry Prize, 668
Davidson, William, of Aberdeen, biography by J. Read (C. Singer) (Nova et Vetera), 380
DAVIES, D. F.: Paralaryngeal actinomycosis in a child, 363
DAVIES, D. R. *See* ALDRIDGE, W. N., 945
DAVIES, E. B.: Technique of shock therapy, 329
— Behaviour problem of children, 1189
DAVIS, D.: The urge to work, 815
DAVSON, H.: *Textbook of General Physiology*, 364
DAWKINS, M.: Xylocaine, 489
DAWSON, Betty, and ZINNEMANN, K.: Incidence and type distribution of capsulated H. influenzæ strains, 740 (O)
DAWSON, E. C.: Attempted suicide, 761
DAYNES, G. *See* HEATON, Nell, 960
DEAFNESS: *See also* Ear; Hearing; Oto-rhino-laryngology
Deaf-mutism, inheritance of, 830
Due to dihydrostreptomycin, 796, 1076 (A)
Further observations on diagnosis in young children, with particular reference to the making of recommendations for special educational treatment (M. R. Dix and C. S. Hallpike), 235 (O); 264 (L)
Decamethonium iodide. *See* Methonium compounds
DEAN, R. F. A.: Development of African children, 159
Death after drowning and electrocution, signs of, 1144
— causes in 1711, 357
— determination of, 275
— medical certification of cause of, 641 (L)
Defamation (Amendment) Bill, 255 (A)
Dehydration; pre- and post-operative treatment, 48
DELAY, J.: *Méthodes Biologiques en Clinique Psychiatrique*, 960
Delirium tremens, endocrine therapy of, 614
Dementia Praecox, or the Group of Schizophrenias (E. Bleuler), 32, 326
DENNISON, W. M.: Congenital absence of rectum, 1084
DENTISTRY: *See also* Caries, dental; Teeth
Dental auxiliaries, 609, 1039
Dental disease among 14-year-old London schoolchildren in 1947 and 1950 (Lady May Mellanby), 7 (O)
Dental services, expenditure on, 760
Dentists' Bill; second reading in the Lords (parliamentary notes), 390; Lords' amendments, 609, 718
Deoxycortone acetate, effect on capillary resistance, 996
Depression, involutional, 1303
DERBES, V. J., and WEISS, T. E.: *Untoward Reactions of Cortisone and A.C.T.H.*, 1072
Dermal sinus, congenital, associated with meningitis, 221
Dermatitis after using "pyrotenex" (C. W. Ward), 905, 1249 (C)
— herpetiformis, in pregnancy, 230
— industrial (parliamentary note), 665
Dermatology: *Therapy of Dermatologic Disorders, including a Guide to Diagnosis and a Dermatologic Pharmacopeia* (S. M. Peck and G. Klein), 1011
— *see also* Skin
Dermatoses, non-urticarial, antihistamines in (P. Inman and I. C. Cowan), 1064 (O)
DEVITT, D. C., NICHOLSON, J. C., and VETTEN, K. B.: Preventing severe surgical shock with A.C.T.H., 1303
Devon and Exeter Medico-Chirurgical Society, 650, 722
DEWHURST, C. J., and ROWLEY, H. A.: Intra-partum uterine rupture, 973
DIABETES MELLITUS: *See also* Insulin
Duration of action of different insulins (F. Gerritzen), 249 (O); 440 (C)
Gangrene treated with α-tocopherol, 276 (correction, 493)
In surgical patients (C. F. Rolland), 737 (O)
Insulin, syringe gauge for (J. C. Page and R. Salt), 1129
International Diabetes Federation Congress, 115, 284
Is bread good for diabetics? 558 (correction, 830)
Pregnancy complicated by diabetes mellitus, conduct of (H. H. F. Barns and M. E. Morgans), 1058, 1248 (C)
Two cases in infants under 1 year (H. S. Brodribb, J. McMurray and L. G. Scott), 1060 (O); 1354 (C)
Diagnostic unit for practitioners, 218
Diaminodiphenysulphone treatment of pulmonary tuberculosis, 1224
Diaminopyridines in treatment of malaria, 730, 732
Diaphragm, congenital hernia of, in siblings (E. E. Philipp and M. O. Skelton), 1283 (O)
Diathermy hæmostat, insulated, 1356
DIBLE, J. H. (editor): *Recent Advances in Bacteriology*. 3rd edition by J. D. Maclennan, 585
DICK, J. C. *See* FINN, O. A., 1067
DICKIE, A. W.: Tuberculosis in families with B.C.G.-vaccinated and non-vaccinated children, 682 (O); 1194 (C)
DICKINSON, C. J.: *Clinical Pathological Data*, 311
Dicoumarol-induced hypoprothrombinæmia, effect of vitamin-K preparations on, 412
Dictionaries: *Black's Medical Dictionary*, 696
— *German–English, English–German Dictionary for Physicians*. Vol. 1: German-English, 1118
"Dieldrin," 543
Digitalis in toxic myocarditis, 1205
Dihydrostreptomycin. *See under* Streptomycin
Di-iodofluorescein in diagnosis of cerebral tumours, 428, 549 (C)
DILLON, F.: The cleft in man, 653
Dimercaprol therapy in mercury nephrosis, 358
Dimethyl phthalate as leech repellent, 898
Dinitro-*ortho*-cresol poisoning, 1025
DINSDALE, T.: Aid to analgesia of the larynx and trachea (preparations and appliances), 707
Diphtheria at Darwen, Lancs (vital statistics), 826
— graphs (vital statistics), 170, 774, 827, 876
— immunization, 268
— — in 1950 (vital statistics), 499
— vital statistics, 774
Disabled, World Federation for, 667
Disabled Citizens (Joan S. Clarke), 1071
DISCOMBE, G.: Agranulocytosis caused by amidopyrine; an avoidable cause of death, 1270 (O); 1292 (A)
Dish-washing with liquor sulphestolis, 724
Disproportion; accuracy of *x*-ray cephalometry *in utero*, 1026
Divorce for incurable insanity (medico-legal note), 52
— Royal Commission on Marriage and Divorce; oral evidence on behalf of B.M.A., 1242; 1237 (L)
DIX, M. R., and HALLPIKE, C. S.: Further observations upon the diagnosis of deafness in young children with particular reference to the making of recommendations for special educational treatment, 235 (O); 264 (L)
DOBRINER, Konrad, obituary of, 715
Doctor. *See* Medical practitioner
Dodds, E. C., awarded Harben Medal, 1142
— *See* COPEMAN, W. S. C., 397
DODDS, G. E.: Blindness in Southern Nigeria, 584; 1248 (C)
DODGSON, Robert William, obituary of, 871
DONALDSON, M.: Educating the public about cancer, 656, 1301
— Barrier of silence, 1195
DOUGLAS, A. S., and BROWN, A.: Effect of vitamin K preparations on hypoprothrombinaemia induced by dicoumarol and "tromexan," 412 (O); 603 (C), 763 (C)
DOUST, J. W. L., and SCHNEIDER, R. A.: Studies on the physiology of awareness; anoxia and the levels of sleep, 449 (O); 475 (L); (correspondence), 971
DOVEY, Ruth: Family planning, 1354
DOWNIE, V. J.: Pulmonary embolism, 1303
DRABKIN, I. E. (Editor): *Caelius Aurellanus: On Acute Diseases and on Chronic Diseases* (C. Singer), 433
DREW, W. R. M., Iraqi Order conferred on, 393
Drinking-water, storage of, 724
Drivers, selection of, 150 (A)
DRUGS:
Bacterial resistance to chemotherapy, 425 (L); 546 (C), 712 (C)
Convictions under Dangerous Drugs Act, 773
Dangerous drugs in the anaesthetic room; legal and practical requirements for recording, 1365
— television programme, 877
Dangerous Drugs Act 1951, 115; reconstitution of tribunal, 824
Prices in advertisements to medical practitioners (parliamentary note), 1361
Proprietary drugs and *National Formulary* equivalents (parliamentary note), 1361
Resistance of micro-organisms to, 434
Samples by registered post, 110
Sensitivity; *p*-aminosalicylic acid, acquired sensitivity to (S. J. Steel), 415
— relation between sensitivity to thiopentone, sulphonamides, and sunlight (F. Evans and J. Gould), 417 (O); 477 (A)
Survey of 17,301 prescriptions on Form E.C. 10 (D. M. Dunlop), 292 (O); 312 (L); (correspondence) 435, 484, 545, 599, 709
Drunkenness among car drivers; trial by magistrate and jury compared, 161
Dublin University. *See* Universities: Dublin
DUNDEE, J. W.: L-*Nor*adrenaline as vasoconstrictor, 547
DUNDON, John, obituary of, 663
DUNLOP, D. M.: Survey of 17,301 prescriptions on Form E.C. 10, 292 (O); 312 (L); (correspondence), 484, 545, 599, 709, 713
DUNLOP, E., and HOSFORD, J.: Case of prostatic abscess, 1392
DUNLOP, James (Glasgow), obituary of, 822
DUNN, H. G.: Acute infectious lymphocytosis, report on a group of cases in a day nursery, 78 (O); 96 (A); 324 (C)
Duodenal juice, trypsin estimation in (I. Gordon, B. Levin, and T. P. Whitehead), 463 (O)
Duodenum; congenital obstruction and mongolism (M. Bodian and others), 77 (O); (correspondence), 276, 384.
— ulcer. *See* Peptic ulcer
Dupuytren's contracture treated with vitamin E (H. J. Richards), 1328 (O)
Durham University. *See* Universities: Durham
DURNIN, J. V. G. A., and WEIR, J. B. DE V.: Statures of a group of university students and of their parents, 1006 (O); (correspondence), 1133, 1300
DUTHIE, J. J. R.: Rheumatic diseases in the U.S.A., 341 (O); 603 (C)
— *See* ROBSON, H. N., 994
DUTTON, Edward Charles, obituary of, 1359
Dyestuff workers, bladder tumours in, 1123 (A)
Dysentery: Absolute viability in water of *Salm. typhi* and the dysentery bacilli (J. F. D. Shrewsbury and G. J. Barson), 954 (O)
— bacillary, epidemiology of human case due to infection by *Sh. flexneri* 103 Z (K. Patricia Carpenter and B. R. Sandiford), 142 (O)
— graphs (vital statistics), 54, 114, 171, 445, 498, 553, 610, 928, 1308, 1363, 1415
Dyskinesia, 1126
Dysmenorrhœa, ovarian, treated by ovarian denervation (D. C. Wiseman and W. M. Thomas), 141 (O); (correspondence), 272
Dysphagia in carcinoma of stomach, 288

Dyspnoea of exertion; test for measurement (P. Hugh-Jones), 65 (O); 95 (A)
Dystrophia myotonica, radiological changes in skull in (J. E. Caughey), 137 (O)

E

EADIE, J.: Period of gestation, 1133
Eales's disease treated with streptomycin, 121
EAR: *See also* Deafness; Hearing; Oto-rhino-laryngology
Ear, Nose, and Throat Diseases for the General Practitioner (W. McKenzie), 473
Otitis media, flying and bathing in, 777
Protruding ears; optimum age for operative correction, 724
Removal of cerumen by water pistol, 223
EAST, Sir Norwood: Criminal responsibility, 45
East Africa: Campaign for blind persons, 393
EBAN, I.: Staining of brain tumours, 549
EBBETTS, J.: Hazards of manipulation, 655
Eclampsia treated with sodium-containing barbiturates, 1091 (correction, 1144)
Economics and doctors, 642 (L); (correspondence), 818
— Coal and cortisone; man-power in a sick society (Ff. Roberts), 321; 437 (C)
Ectopia cordis; case surviving nine hours (Eleanor J. Wilson), 1175; 1358 (C)
Eczema, infantile; treatment with goat's milk, 709, 973
— of hand in child, treatment of, 1312, 1418
EDGE, J. R.: Pulmonary tuberculosis and pregnancy, 845 (O); 862 (A); (correspondence), 1030
EDHOLM, O. G.: Tropical fatigue, 815
Edinburgh University. *See* Universities: Edinburgh
Edinburgh University Club of London, 1415
EDINGTON, G. M.: Sickle-cell trait in the newborn, 763
Education: Higher grants for students, 1415
— medical: Care of the neonate; instruction of the medical undergraduate (W. S. Craig), 1062 (O)
— — full-time teachers without experience in private practice, 974
— postgraduate; Government scheme for refresher courses, 55
— schools for the educationally subnormal, 286
EDWARDS, H. C.: Diseases of the gall-bladder, 1125
EDWARDS, P. W.: *Isonicotinic hydrazide as wonder drug*, 868
— and MIDGLEY, R. L.: Shortage of *x*-ray film, 49
— PENMAN, A. C., and CUTBILL, L. J.: Sulphone and streptomycin in pulmonary tuberculosis, 1224 (O)
EGERTON, Mary E.: Two cases of tetanus neonatorum treated with penicillin and aureomycin, 470
Egypt: Health of British troops, 997
EICKEN, C. VON, and TREECK, A. S. VAN: *Atlas der Hals-, Nasen-, Ohren-Krankheiten*, 3rd enlarged ed., 1072
Eire. *See* Ireland, Republic of
Electric convulsion therapy; succinylcholine as relaxant in electro-shock, 1027
— — — technique for prevention of injuries, 109, 329
— — — use of hexamethonium bromide and decamethonium iodide with (J. D. Montagu), 1336
Electrical conductor ("pyrotenex"); dermatitis after use (C. W. Ward), 905; 1249 (C)
— injuries: *Fortschritte in der Behandlung schwerer und schwerster Hochspannungsunfälle* (H. Fischer and R. Fröhlicher), 203
Electrocardiography: *Clinical Unipolar Electrocardiography* (B. S. Lipman and E. Massie), 1287
Electronic brain, 542
ELFORD, William Joseph, obituary of, 495
Eli Lilly travelling fellowships in medicine, 445
Elizabeth II, accession of, 367 (L)
Birthday Honours List, 1299
Honorary physicians to, 983
Loyal Address of B.M.A., 482
Patron of B.M.A., 1340 (L)
ELKAN, E.: Sexual disorders in women, 105
— Ectopia cordis in toads, 1358
ELLIOTT, Edith M. W.: Pseudocyesis, 657
ELLIS, A. S.: Ethics of leucotomy, 1131
ELLIS, F. R.: Tropical fatigue, 815
ELLIS, G. R. *See* LITTLER, T. R., 842
ELLISON, N. P., and CHAMBERS, J. S. W.: Torsion of Fallopian tube during pregnancy, 694; 1251 (C)
ELLISON, Philip Oswald, obituary of, 871
ELMES, B. G. T.: Where do snails go? 820
Embolism, air, pathogenesis of, 892
— — post-mortem diagnosis by radiography (J. D. Taylor), 890 (O)
— and thrombosis of cerebral vessels; treatment (R. Leriche), 231 (O); 262 (L); 381 (C), 486 (C), 487 (C); 777
— prolonged anticoagulant therapy with heparin, (A. Wynn, J. F. Goodwin, and Angela Birbeck), 893 (O)
— pulmonary; survey in a general hospital (D. S. Short), 790 (O); 809 (A); (correspondence), 920, 1303, 1405
Emergency Bed Service; applications and admissions, 56, 114, 228, 283, 338, 394, 446, 500, 555, 612, 668, 722, 776, 828, 877, 929, 983, 1039, 1090, 1142; tribute to, 1304
Emetine in scorpion stings, 396, 614
EMMENS, C. W. (Editor): *Hormone Assay*, 424
EMMETT, J. L. *See* BRAASCH, W. F., 1011
Emphysema and associated dyspepsia, 220, 655
Empire Rheumatism Council; fifteenth annual meeting, 1024
Empyema; pathology, diagnosis, and treatment (T. H. Sellors), 704
Encephalitis: Outbreak possibly due to poliomyelitis virus (A. M. Barrett, D. Gairdner, and A. M. McFarlan), 1317 (O)
Enderlein's laboratory closed, 929
Endocarditis, bacterial, caused by coagulase-negative *Staph. albus* (C. C. Cobb), 90
Endocrines: Hyperfunction, 729
Endocrinology: *Annual Report on Stress* (H. Selye), 1288
— *Diseases of the Endocrine Glands* (L. J. Soffer), 748
— Recent advances (S. L. Simpson), 725 (O)
Endometriosis, hormone therapy in, 286
ENGEL, D.: Cerebral blood vessels and "priscol," 106
England and Wales: Registrar-General's quarterly returns (vital statistics), 982
Enteric fever: Graphs (vital statistics), 170, 666, 721, 981, 1038, 1088, 1141, 1202, 1256, 1309, 1362, 1414
— — vaccination, 270
Enterogastrone in treatment of peptic ulcer, 109
Enuresis, 547
Enzyme at sympathetic nerve endings (J. H. Burn), 784 (O); 861 (A)
Enzymes. Chemistry and Mechanism of Action (Editors: J. B. Sumner and K. Myrbäck), 423
Eosinophil level in relation to adrenal cortical function (P. B. Jennings), 1055 (O); 1249 (C)
Epidemiological notes. *See* Vital statistics
Epidemiology: *Global Epidemiology: A Geography of Disease and Sanitation*. Vol. 2: *Africa and the Adjacent Islands* (J. S. Simmons and others), 1010
Epilepsy, laryngeal, 653, 1250
— psychomotor, diagnosis of, 395
— spontaneous arterial occlusion during fit (W. L. B. Leese), 855; 1303 (C)
— value of pneumoencephalography in diagnosis (J. Marshall and C. W. M. Whitty), 847 (O); 1194 (C)
Epileptic car drivers, 489
Epileptics, employment of, 324
— management of, 323
— schools for (parliamentary notes), 391
Epistaxis, 1124 (A); 1197 (C)
Ergonomics Research Society: Symposium on fatigue, 805 (L); 814
Erythema nodosum, familial (J. Fry), 529 (O); (correspondence), 766
Erythroblastosis foetalis, 108
— — icterus gravis and cirrhosis of liver, 1385
— — study of the A factor as a cause of haemolytic disease in premature babies (S. Phansomboon and O. J. Pollak), 1170 (O)
Erythrocytes, effect of hypochromasia on specific gravity and sedimentation of (K. B. Rogers), 1109 (O) (correction, 1418); 1407 (C)
— sedimentation rate; anomaly in rheumatic diseases (Sylvia Goldberg, L. E. Glynn, and E. G. L. Bywaters), 202
— — — correction in anaemia; experimental study based on interchange of cells and plasma between normal and anaemic subjects (J. C. F. Poole and G. A. C. Summers), 353 (O); 369 (A)
— — — effect of cortisone on 402
— — — Westergren and Wintrobe methods compared, 277
ESPLEN, J. R.: Technique of shock therapy, 109
Ethics: *Ethical Basis of Medical Practice* (W. L. Sperry), 1118
— doctors' opinions in lay press, 599
Ethyl acetate, toxic effects, 174
Ethyl chloride for unconscious hysterics, 1191
Ethylene diamine tartrate crystals, effects of contact with, 1418
EUNSON, L. H.: Isonicotinic hydrazide as wonder drug, 970
EVANS, F.: Ryle's tube in trachea, 920
— Needle biopsy of liver, 1249
— and GOULD, J.: Relation between sensitivity to thiopentone, sulphonamides, and sunlight, 417 (O); 477 (A)
EVANS, G.: Inaugural Langdon-Brown Lecture, 175 (O); 206 (L)
EVANS, Rees, "cancer cure," 824; report of investigating committee, 1236 (L); 1253 (parliamentary note)
Everest expedition, 1120 (L)
Evidence, expert, 644 (A)
Exchange Control Medical Advisory Committee, 1081
Exchange transfusion. *See* Blood transfusion
Exercise; simple standard test and its use for measuring exertion dyspnoea (P. Hugh-Jones), 65 (O); 95 (A)
Exophthalmos, 49
Experimental Parasitology, 172
EYE: *See also* Blindness; Colour vision; Cornea; Lens; Ophthalmology; Vision
Accommodation in ophthalmoscopy, 1042
Artificial-eye specialists, 283
Eyestrain, causes and effects, 229
Foreign body, removal of, 975
Glaucomas (H. S. Sugar), 586
Intra-ocular acrylic lenses, 1121 (A)
The Significance of Heredity in Ophthalmology. A Tasmanian Survey (J. B. Hamilton), 803
EYE (*continued*):
Styes, recurrent, prevention, 1259
Surgery of the Oblique Muscles of the Eye (W. H. Fink), 747
Systemic Ophthalmology (Editor: A. Sorsby), 310
Tolerance to streptomycin, 119
Tumors of the Eye (A. B. Reese), 638

F

Factories; colour coding of danger points, 675
Faculty of Anaesthetists, 168; fourth annual general meeting, 825
Faculty of Ophthalmologists: Officers elected, 1411
Faculty of Radiologists: Fellows elected, 281
FAIRFIELD, Letitia: Family planning, 708
— *See* FRANKLIN, A. W., 161
FALKNER, F.: Growth and development of children, 1300
Fallopian tube, torsion; case (P. G. Seed), 1176; 1407 (C)
— — torsion during pregnancy (N. P. Ellison and J. S. W. Chambers), 694; 1251 (C)
— — tuberculosis of, treated with *iso*nicotinic hydrazide, 1028
— — tubo-uterine implantation (V. B. Green-Armytage), 1222 (O); 1355 (C)
Family Doctor, 39 (A)
Family planning; analysis of letters to David Mace, 866
— — with particular reference to contraception; memorandum prepared by Medical Women's Federation, 595; (correspondence), 708, 766, 867, 970, 1027, 1132, 1247, 1354
Family research at Peckham, 317 (A); 490 (C)
Fantus estimation of urinary chloride, 275
Fat emulsions, intravenous administration of, 1400 (A)
— intolerance, treatment of, 1196
Fatigue; symposium of Ergonomics Research Society, 805 (L); 814
FEDDON, Walter Fedde, obituary of, 662
FEENY, P. J. *See* THOMAS, J. M., 1249
Fees: Private patient's fees (medico-legal note), 551
Feet. *See* Foot
FEIWEL, M.: Plantar warts in children, 712
FELL, Honor, elected F.R.S., 703
Femur: Acrylic replacement after segmental resection (J. M. P. Clark and A. C. Bingold), 903 (O)
— treatment of recent transcervical fractures of (D. O. Williams and E. K. McLean), 580 (O); 592 (A); (correspondence), 764
FERGUSON, Joshua, obituary of, 167
FERGUSON, V. L.: Latin comes to life, 1304
FERGUSSON, A. G., MCINTYRE, J. P., and GEMMELL, A. R.: Toxic hepatitis and P.A.S., 855
FERGUSSON, J. D.: Sulphamerazine still dangerous, 713
FERNANDO, H. V. J. *See* ABBOTT, J. D., 523
Ferrous sulphate, effect on "physiological" anaemia of pregnancy (Nancy Benstead and G. W. Theobald), 407 (O); 476 (A); 599 (C), 817 (C)
— — poisoning in children, liver damage in, 438
FERTILITY and INFERTILITY:
Fertility after prostatectomy, 670
Human Fertility: The Modern Dilemma (R. C. Cook), 145
Infertility in the male, 106, 218, 328, 656
International association formed, 1203
Treatment of infertility by tubo-uterine implantation (V. B. Green-Armytage), 1222 (O); 153 (C)
FIELDING, G.: Redheads, 1260
Filariasis: Investigations into the cause of microfilarial periodicity (J. A. McFadzean), 1106 (O)
FILMS:
B.M.A. film catalogue, 152
Cheap method for recording sound, 1173
Medical films in Spain, 568
Prizes for medical films, 393, 983
S.F.A. Catalogue of Medical Films, 1257
FINDLAY, George William Marshall, obituary of, 658, 715, 769, 822
Finger: Paronychia, chronic, treatment, 58
Fingernails, white spots on, 985
Finger-print development, chronic mercury poisoning due to (G. Forbes and J. White), 899 (O); (correspondence), 1131, 1406
FINK, W. H.: *Surgery of the Oblique Muscles of the Eye*, 747
FINLAY, H. V. L., and BOUND, J. P.: Neonatal urticaria, 1134
FINN, O. A., DICK, J. C., and STEVENSON, J. S.: Case of accidental generalized vaccinia, 1067 (O); (correspondence), 1134, 1249
Fire extinguishers, poison in, 1122 (A)
FISCHER, H., and FRÖHLICHER, R.: *Fortschritte in der Behandlung schwerer und schwerster Hochspannungsunfälle*, 203
Fits for diagnosis, 395
— from African worms, 339
FITZGERALD, D. P.: Emergency Bed Service, 1304
FITZGERALD, F. P.: Nailing the femoral neck, 764
FITZGERALD, O.: Discharge of certified mental patients, 1082
FITZGERALD, Ruth: Social trends and home confinements, 1353

FITZGERALD, T. B.: Habitual abortion, 819
"Fixanol C," use in sterilization of hospital blankets, 22
FLANAGAN, H. M.: Ethics of leucotomy, 1132
"Flaxedil." *See* Gallamine triethiodide
FLEISCHHACKER, H. H.: Chilblains and hypothyroidism, 711
FLEMING, G. W.: School medical service, 490
FLEMING, Geoffrey Balmanno, obituary of, 977
FLETCHER, Nigel Corbet, obituary of, 223
Flexor pollicis longus, teno-vaginitis of, 199
Flour, effect of agene on vitamins in, 614
Fluids, surgical; recommended methods of colouring, 982
Fluorescent lighting, position in drawing-office, 230
Foetus: Growth of biparietal diameter of head in last weeks of pregnancy (I. MacDonald), 798 (O); (correspondence), 922, 1408
FOLDES, F. F.: Prolonged respiratory paralysis after succinylcholine, 1352
Folic acid added to iron pills, 599
— — in megaloblastic anaemia of infancy, 1226
Folliculitis, epidemic, at Cheltenham, 1203, 1303

FOOD: *See also* Nutrition; Vitamins
Copper content, 1193
Lead as contaminant, 218
The Malthusian Controversy (K. Smith), 907; 1134 (C)
Statutory requirements for sanitary conditions of sale (parliamentary note), 1200
Supply: *Four Thousand Million Mouths* (Editors: F. LeG. Clark and N. W. Pirie), 856

Food-poisoning (Joan Taylor), 593
— Ministry of Health portable exhibition, 844
— *Salm. minnesota* infection, 38 (A); 53 (vital statistics)
— Staphylococcal mastitis in cows as cause, 1144
Foot: Gangrene of unknown aetiology (M. G. Philpott), 201
— National Foot Health Week, 667
— trauma and fibrosarcoma in (E. Silverstone), 958
— warts; diagnosis and treatment (L. Forman), 480; 712 (C)
Foot-and-mouth disease (parliamentary note), 1087
— — insufficient research on, 1251
FORBES, G., and WHITE, J.: Chronic mercury poisoning in latent finger-print development, 899 (O); (correspondence), 1131, 1406
FORBES, R.: Unsuitable advice, 440
— Anatomy Act, 651
FORBES, William John, obituary of, 1305
FORD, C. S., and BEACH, F. A.: *Patterns of Sexual Behavior*, 1338
Forensic medicine. *See* Medico-legal
FORMAN, L.: Warts of the feet, 480; 712 (C)
— Familial erythema nodosum, 766
Formol gel reaction, discovery of, 163
Formosa: Malaria eradication, 1141
FORRESTER-BROWN, Maud F.: "Idiopathic" scoliosis, 219
— Early treatment of poliomyelitis, 923
FORSHALL, Isabella: Congenital absence of rectum, 762
FORSTER, A.: What doctors prescribe, 545
FOSBERY, W.: Seasickness, 1042
— Debauchery of honest words, 1084
FOSTER, J.: Common causes of curable blindness, 318; 490 (C), 651 (C), 762 (C), 1083 (C)
FOX, E. O.: Shortage of *x*-ray films, 219
FOY, H., KONDI, A., and HARGREAVES, A.: Response of the megaloblastic anaemias of pregnancy to animal protein factor; preliminary report, 852 (O)
Fractures in the aged (R. H. Metcalfe), 1240 (correction, 1366)
France: Anglo-French medical exchange bursaries, 555, 1309
— Scholarships in medical science, 227
FRANKLIN, A. W.: Incontinentia pigmenti; report of case, with summary of 29 cases from the literature, 75 (O); (correspondence), 277, 439
— FAIRFIELD, Letitia, and RATTENBURY, G.: Homes for incurable children, 161
FRANKLIN, L. M.: Peacemaking, 1031
— TEPPER, R., PAULETT, J. D., and TUCKMAN, E.: What doctors prescribe, 485
FRANKLIN, Olga: *Born Twice*, 1011
FRANKLIN, R. H., and SHIPMAN, J. J.: Carcinoma of oesophagus; diagnosis and surgical treatment of 159 cases, 947 (O); 961 (L)
FRASER, D. E. *See* HOBSON, A. C. S., 1111
FRASER, Robert Moore, obituary of, 496
FREEMAN, D. M. *See* CHAMBERLAIN, E. N., 1145
FREUDENTHAL, Walter, obituary of, 820
FRIEDMANN, I.: Malignant granuloma of the nose, 491
FRIEND, J., and THACKRAY, A. C.: Atypical abdominal tuberculosis with haematological manifestations, 574 (O)
FROEHLICH, A. L.: *Xanthomatoses*, 695
FRÖHLICHER, R. *See* FISCHER, H., 203
FRY, J.: Familial erythema nodosum, 529 (O); (correspondence), 766
FULTON, J. F.: *Frontal Lobotomy and Affective Behavior: A Neurophysiological Analysis*, 1394
Fungus infections: Organization for medical mycology, 1400 (A)
— — Pulmonary aspergillosis following antibiotic treatment of post-influenzal bronchopneumonia (J. D. Abbott and others), 523 (O); 537 (L)
Funnel chest, 370 (A); 715 (C)

G

GABB, H.: Educating the public about cancer, 656
GABRIEL, W. B.: Surgical treatment of chronic ulcerative colitis, 881 (O); 910 (L); 1194 (C)
GAIRDNER, D. *See* BARRETT, A. M., 1317
Galactosaemia, amino-aciduria in (A. Holzel, G. M. Komrower, and V. K. Wilson), 194 (O)
GALE, E. F.: Drug resistance of micro-organisms, 434
Galen: *Translation of Galen's Hygiene* (*De Sanitate Tuenda*) by R. M. Green (Nova et Vetera) (C. Singer), 1404
Gallamine triethiodide, disadvantages in electric convulsion therapy, 109
Gall-bladder, diseases of (H. C. Edwards), 1125
Gall-stones, 1125
— clinical survey (T. R. Littler and G. R. Ellis), 842 (O); 1031 (C)
GALPINE, J. F., and WALKOWSKI, J.: Case of mumps with involvement of the lacrimal glands, 1069; 1250 (C)
GAMGEE, L.: Meigs's syndrome and Lawson Tait, 1031
GAMMON, Julia. *See* GREENE, R., 17
Ganglion block, autonomic, during anaesthesia, 1144
Gangrene of unknown aetiology in the foot (M. G. Philpott), 201
— progressive bacterial synergic, of legs (R. Marcus), 1230
GARAI, O.: Parkinsonism, 382
GARDINER, P. A.: Death on the roads, 108
GARDNER, A. D.: Debauchery of honest words, 715
GARDNER, Isabel M.: Family planning, 1247
GARLAND, H. G.: Parkinsonism, 153; 382 (C), 490 (C)
GARLAND, T. O.: Dead or alive?, 275
GARNHAM, P. C. C., appointed to Chair of Medical Protozoology, London School of Hygiene and Tropical Medicine, 1254
GARROD, L. P.: Drug resistance of micro-organisms, 434
— Sensitivity of *Actinomyces israeli* to antibiotics, 1263 (O)
GASKELL, Sir Arthur, obituary of, 278
GASSER, C.: *Hämolytische Syndrome im Kindesalter*, 857
GASTON, Joseph, obituary of, 822
Gastroenteritis: "Epidemic" abdominal colic associated with steatorrhoea (M. E. M. Thomas), 691 (O)
Gaucher's disease, splenogram in, 989
GEAR, J. H. S., awarded James D. Bruce Memorial Lectureship and Medal, 1090
GEIRINGER, E.: Treatment of antihisan poisoning, 163
GELFAND, M., and BARNETT, E. E.: A peculiar syndrome of bladder paralysis in natives of South Central Africa, 1172 (O); 1408 (C)
GEMMELL, A. R. *See* FERGUSSON, A. G., 855
General Medical Services Committee report on remuneration, 1235

GENERAL PRACTICE: *See also* Medical practitioner
College of General Practice, 1410
Fouling the nest, 974
General practitioners, closer link with hospital services, 151 (A)
— — coronary disease among, 503, 537
— — in hospitals (parliamentary notes), 770, 1087
— — refresher courses for, 55
Remuneration; preparing the case for adjudication, 263 (L); adjudication, 697 (L), 749; 718, 771; 1253 (parliamentary notes); working party, 1235 (L)
— report of General Medical Services Committee, 1235
— White Paper on terms of reference and recommendations of Working Party suggested (parliamentary note), 1200
Restrictive covenant appeal dismissed (medico-legal note), 111

Genetics: *Genetics in Ophthalmology* (A. Sorsby), 203
— *Genetics of Micro-Organisms* (D. G. Catcheside), 907
— *Soviet Genetics* (A. G. Morton), 472; 708 (C)
George VI, death of, 366 (L); 386 (correction, 549)
Geriatrics. *See* Old Age
Germany: Present state of medicine, 584
Gerontology. *See* Old age
GERRARD, J.: Icterus gravis and cirrhosis of the liver, 1385 (O)
GERRITZEN, F.: Duration of the action of different insulins, 249 (O); 440 (C)
Gestation. *See* Pregnancy
GHOSE, R.: L-*Nor*adrenaline as a vasoconstrictor, 385
Giardia lamblia infestation, relationship to acute infectious lymphocytosis, 79
GIBB, W. E. *See* STORY, P., 990
GIBBENS, J. H.: Character training of children, 1189
GIBBENS, T.: Medico-legal aspects of suicide, 1130
GIBBS, J. R. *See* OLIVER, L. C., 634
GIBSON, H. L.: *Photography of Patients*, 1394
GIBSON, J. R. *See* MCKEOWN, T., 938
GIFFORD, J. H. *See* LEGGAT, P. O., 1008
GILBERT, I.: Angioma venosum racemosum with angiomatous lesions of skin and omentum, 468 (O)
GILLHESPY, R. O.: Parkinsonism, 382
GILLIES, H., HICKSON, Barbara, and MAYER-GROSS, W.: Follow-up study of 238 leucotomized patients, 527 (O); 909 (L)
GINSBURG, Ethel L.: *Public Health is People*, 857
GIRDWOOD, R. H.: Treatment of anaemia, 599
Girls, over-sexed, hormone treatment of, 986
Glandular fever; report of 5 cases (J. F. Goodall), 636
— — unusual features in fatal case (S. Marshall and K. S. Millingen), 1325 (O)
— — with cerebral complications; case (I. M. Librach), 956 (O)
GLANVILL, A. T. H., and MARTYR, B.: Interesting tumour of palate, 329
Glasgow; influenza outbreak (vital statistics), 720
— University. *See* Universities: Glasgow
Glasgow Royal Cancer Hospital changes name, 983
Glaucoma, congestive and non-congestive, 319
— congestive and simple, symptomatology of (S. J. H. Miller), 456 (O); 712 (C), 764 (C), 869 (C), 1083 (C), 1356 (C)
Glaucomas (H. S. Sugar), 586
GLEAVE, Humphrey Hart, obituary of, 925
GLEDHILL, R. C., and SHILLITOE, A. J.: Purpura and amyloidosis in Hodgkin's disease, 1336
GLENNIE, J. S., DARK, J. F., and LINDARS, D. C.: Unsuspected foreign body in putrid bronchiectasis, 1009
Gluteal evolution; *Cold Spring Harbor Symposia on Quantitative Biology*, Vol. XV, 260
GLYN, J. H. H. *See* COPEMAN, W. S. C., 397
GLYNN, L. E. *See* GOLDBERG, Sylvia, 202
Goat's milk in treatment of infantile eczema, 709, 973
GODDING, E. W.: Mild aperient for the elderly, 830
GOLD, H., and others (Editors): *Cornell Conferences on Therapy*, 205
GOLDBERG, Sylvia, GLYNN, L. E., and BYWATERS, E. G. L.: Anomaly of sedimentation rate in rheumatic diseases, 202
GOLDEN, G. N.: Hip reconstruction by acrylic prosthesis, 48
Gonadotrophin, chorionic, assay in pregnancy, 621
Gonorrhoea, treatment of (T. E. Osmond), 863
GOOD, I. J.: Foreign body in the eye, 975
GOODALL, J. F.: Glandular fever, 636
GOODWIN, J. F. *See* WYNN, A., 893
GOODWIN, L. G.: "Daraprim" (B.W. 50–63); new antimalarial; trials in human volunteers, 732 (O), (correction, 870); 1075 (A)
GORDON, I., LEVIN, B., and WHITEHEAD, T. P.: Estimation of trypsin in duodenal juice, 463 (O)
GORDON, M. H.: Accidental generalized vaccinia, 1134
— eightieth birthday of, 1350
GORDON, Thomas Landles, obituary of, 52
Gordon-Taylor, Sir Gordon, Sc.D.hon.Cantab., conferred on, 1293 (A)
GORNALL, R. G.: Action of different insulins, 440
Gorilla: *Anatomy of the Gorilla* (W. B. Atkinson and others), 365
GOSSE, P.: *Dr. Viper. The Querulous Life of Philip Thicknesse*, 1338
GOSSIP, J.: Death on the roads, 161
GOUGH-THOMAS, John. *See* RAVEN, R. W., 381
GOULD, I. *See* EVANS, F., 417
GOULD, R. B.: Succinylcholine chloride, 440
Goulstonian Lectures, 1093, 1152, 1260 (correction)
Gout, albuminuria in, 670
— and insulin hypoglycaemia, 1251
— pathogenesis of, 346
GOWANS, Francis Jollie, obituary of, 926
GRABER, I. G. *See* SHACKMAN, R., 1284
GRACE, W. J., WOLF, S., and WOLFF, H. G.: *The Human Colon: An Experimental Study based on a Direct Observation of four Fistulous Subjects*, 959
Gräfenberg ring, 1418
GRAF, A. K.: Is Latin dead? 1084
Grafts: Traumatic autografting, 274
— *see also* Cornea; skin
GRAHAM, F. O., and COLEMAN, P. N.: Infection of secondary carcinoma by *salm. montevideo*, 1116
GRAHAM, Frederick Montague, obituary of, 550
GRAHAM, J. D. P.: What doctors prescribe, 435
GRAHAM, J. G. *See* CAMPBELL, A. J. M., 251
GRANT, G.: Prolonged respiratory paralysis after succinylcholine, 1352
GRANT, H. M.: Prostatic obstruction and inguinal hernia, 547
GRANT, J.: Family planning, 970
GRATTAN, Henry William, obituary of, 1411
GRAYSON, J., and JOHNSON, D. H.: L-*Nor*adrenaline as vasoconstrictor, 546
Great Ormond Street, centenary of (T. T. Higgins), 371 (A); 377, 1040; 1184 (A)
Great Ormond Street Journal, 392
Greece: *Nutrition Work in Greece* (A. G. Tsongas), 145
— School of Hygiene, Athens, reorganization of, 808
GREEN, R. M.: *Translation of Galen's Hygiene* (*De Sanitate Tuenda*) (Nova et Vetera) (C. Singer), 1404
GREEN-ARMYTAGE, V. B.: Tubo-uterine implantation, 1222 (O); 1355 (C)
GREENE, R., VAUGHAN-MORGAN, Josephine, and GAMMON, Julia: Effect on bleeding-time of an extract of blood of patients with rheumatoid arthritis, 17 (O); 438 (C), 603 (C)
GREENWOOD, J. M.: Re-education in walking, 1300
GREEVES, R. A.: Congestive and simple glaucoma, 764, 1083
GREGORY, Hazel Howard Chodak, obituary of, 278
GREGORY, J. R.: *Under the Sun. A Memoir of Dr. R. W. Burkitt, of Kenya*, 311
GREGSON, A. H.: What doctors prescribe, 709
GREIG, John Isdale, obituary of, 332
GRENVILLE-MATHERS, R. *See* TRENCHARD, H. J., 1194

GRIEVE, W. S. M. *See* LOCKET, S., 254
GRIFFITHS, D. LL.: Tenosynovitis and tendovaginitis, 645 (correspondence), 766
GRIFOLS-LUCAS, J. A.: Use of plasmapheresis in blood donors, 854
GRIMSHAW, C.: Xylocaine, 489
Growth hormone, 725
— promoters, 640 (L); 1357 (C)
GUERRIER, S. M., and MASON, J. C.: Undesirable side-effects from decamethonium iodide, 1329 (O)
GUEUKDJIAN, S. A.: Continental views on leucotomy, 1192
GUNESEKERA, Sir Frank Arnold, obituary of, 494
GUNTHER, Mavis: Vitamins during lactation, 396
GURLING, K. *See* ABBOTT, J. D., 523
Gynaecology: *Handbook of Gynaecology* (T. L. S. Baynes), 638
— Leukaemia with primary gynaecological manifestations (C. E. W. Wheaton), 30

H

HAAS, M. P. *See* HAAS, S. V., 32
HAAS, S. V., and HAAS, M. P.: *Management of Coeliac Disease*, 32
HABER, H.: Incontinentia pigmenti, 277
HABERMAN, S., and HILL, J. M.: On the verbal usage of the CDE notation for the Rh blood groups, 851 (O)
HADAWAY, A. B.: Physical factors affecting the efficiency of insecticides, 543
HADFIELD, C. F.: Forgotten tourniquet, 328
HADFIELD, G. J. *See* KINMONTH, J. B., 1377
Haematemesis and melaena in carcinoma of stomach, 288
Haematology. *See* Blood
Haemoglobin levels in adults and children (W. T. C. Berry, P. J. Cowin, and H. E. Magee), 410 (O)
Haemoglobinuria: Acute idiopathic paroxysmal cold haemoglobinuria of non-syphilitic type in a child (W. P. Sweetnam, E. F. Murphy, and R. C. Woodcock), 465 (O)
Haemolytic disease in premature babies; study on the A factor as a cause, 1170
— — of newborn, 108
Haemophilia: Dr. W. A. Timperley's method of treatment (parliamentary note), 1413
Haemophilus influenzae, incidence and type distribution of capsulated strains, 740 (O)
Haemorrhage, intracranial, spontaneous, in the young, 1013
— surgical, reduction by preoperative arteriotomy, 965 (A)
Haemorrhagic disease, epidemic, in Korea, 477 (A)
HAFNER, R.: Case illustrating hazards of manipulative treatment in lumbo-sacral disk protrusions, 361 (O); (correspondence), 655
HAINES, R. W. *See* BOCOCK, E. J., 424
Hair, excessive; steroid metabolism in idiopathic hirsutism, 39 (A)
— oil, home-made, 614; 724 (C)
— physiology of, 369 (A)
— red, racial distribution of, 1143, 1260
Haiti: Unesco project for reducing illiteracy and encouraging hygiene and social welfare, 96 (A)
HAKIM, A. E.: Indian remedies for poor memory, 222
Halden's emulsifying base, 670
HALER, D.: Anaemia in pregnancy, 817
HALL, C. L.: Wandering spleen during first decade of life, 957; 1407 (C)
HALL, Charles Herbert, obituary of, 717
HALL, E. G. *See* COHEN, B. E., 48
HALL, Margaret E.: Acute infectious lymphocytosis, 324
HALLAM, J.: Penicillin dangerous? 817
Hallett Prize, 443
HALLINAN, T. C.: Treatment of tropical ulcers, 493
HALLPIKE, C. S. *See* DIX, M. R., 235
HALPERN, B. N.: Mode of action of synthetic antihistaminic drugs, 380
Halsted, W. S., centenary of birth of, 1002
HAMBLING, J.: Epidemiology of hypertension, 1083
— Management of hypertension, 1248
HAMBLY, T.: Tenosynovitis, 766
HAMILTON, D.: Primary atypical pneumonia, 110
HAMILTON, E. M.: Terramycin, 1197
HAMILTON, J. B.: *The Significance of Heredity in Ophthalmology. A Tasmanian Survey*, 803
HANCE, Sir Bennett: Malaria, 104, 492
Hand. *See* Finger; Nails
Handedness: Left hand, right hand, 591 (A); 763 (C)
Handicapped persons: *Summary of Legislation and Directory of Organizations for the Care of the Physically Handicapped*, 195
HANDLEY, R. S.: Is metallic mercury poisonous, 1131
HANDLEY, W. S., eightieth birthday of, 812
HANLON, J. J.: *Principles of Public Health Administration*, 959
HANSCHELL, H. M.: Pulmonary embolism from calf veins, 1405
Harben Medal, 1142
HARDIE, D.: Place for voluntary work, 657
HARDY, R.: Work of health visitors, 1355
HARE, John, obituary of, 1198
HARGREAVES, A. *See* FOY, H., 852
HARKINS, W.: Is Latin dead? 1084
HARLAND, S. C.: Soviet genetics, 708
HARMAN, J. B.: Oesophagitis, 941 (O); 961 (L); 1250 (C)
HARPER, J. K.: Prolonged respiratory paralysis after succinylocholine, 866, 1352
HARRIS, G.: Carcinoma of adrenal cortex, 655
HARRIS, I.: Liverpool Heart Hospital, 1355
HARRIS, L. J.: *Vitamins. A Digest of Current Knowledge*, 310
HARRIS, T. A. B.: *Mode of Action of Anaesthetics*, 423
HARRIS, W.: Fifth and seventh cranial nerves in relation to nervous mechanism of taste sensation; a new approach, 831 (O); 860 (A)
HARRISON, C. M.: Resistance of insects to insecticides, 543
HARRISON, L. W.: Malaria, 220
HARRISON, R. J., and ABELSON, D.: Carcinoma of adrenal cortex with endocrine manifestations; report of a case, 303 (O); (correspondence), 303
HARRISON, T. R., and others (Editors), *Principles of Internal Medicine*, 364
HARRISON, W. R. E.: Radiography and ruin, 219
HART, F. D.: Cortisone and A.C.T.H. in treatment of ankylosing spondylitis, 188 (O)
— WRAITH, D. G., and MANSELL, E. J. B.: Agranulocytosis successfully treated with A.C.T.H., 1273 (O); 1292 (A)
HART, P. D.: Bacteriological warfare in Korea, 652
— LONG, D. A., and REES, R. J. W.: Depression of tuberculin sensitivity in guinea-pigs by certain antituberculous surface-active agents, 680 (O); 702 (A)
HARTMANN, Henri, obituary of, 167
HARTMANN, Max, obituary of, 389
Harveian Lecture, 1367
Harveian Society of London, 55, 1141
HARWOOD, Leonard Austin, obituary of, 1252
HASEEB, M. A.: "Nilodin" in treatment of *Schistosoma haematobium*, 1331 (O)
HASS, J.: *Congenital Dislocation of the Hip*, 1287
HAUGHTON, William Steele, obituary of, 331
HAWKE, A.: Criminal responsibility, 45
HAWKING, F.: Histological study of onchocerciasis treated with hetrazan, 992 (O)
HAWKSWORTH, W.: Present conception of trial labour, 1025
HAY, J. D. *See* CHAMBERLAIN, E. N., 1145
HAY, M. B.: What doctors prescribe, 600
HAYMAN, F. K.: Coronary disease in doctors, 651
Head, foetal, growth of biparietal diameter in last weeks of pregnancy (I. MacDonald), 798 (O); (correspondence), 922, 1408
— injury, cephalic tetanus after (L. Bagratuni), 461 (O)
— size and rate of growth, 1300
Head-dropping test (R. Wartenberg), 687 (O)
HEADY, J. A. *See* MORRIS, J. N., 503
Health visitors, 1077 (A); 1355 (C)
HEANLEY, C.: Skin lesions in ulcerative colitis, 1407
Hearing: *See also* Deafness; Ear; Otorhinolaryngology
— Testing in schoolchildren, 117
Hearing-aids issued, 391

HEART:
Approach to Cardiology (J. C. Bramwell), 907
Artificial heart, 842
Cardiac massage after collapse on the operating table, 1181 (A)
Catheterization in mitral stenosis, 1052
Clinical Unipolar Electrocardiography (B. S. Lipman and E. Massie), 1287
Coronary disease; aetiological relationship of prolonged hyperpiesia, 723
— — in medical practitioners (J. N. Morris, J. A. Heady, and R. G. Barley), 503 (O); 535 (L); (correspondence), 651
Cortisone in rheumatic carditis: Some preliminary observations (E. N. Chamberlain, J. D. Hay, and D. M. Freeman), 1145 (O); 1179 (L)
Doctors and heart disease research, 1190
Ectopia cordis; case surviving nine hours (Eleanor J. Wilson), 1175; 1358 (C)
Failure, left ventricular, acute; relief by "priscol" (D. Wheatley), 1174 (O)
First European Congress of Cardiology, 115
Fourth Interamerican Congress of Cardiology, 338
Heart block (G. Bourne), 966
Liverpool Heart Hospital, 1355
Mitral valve disease in the light of surgical treatment (G. Bourne), 896 (O)
Myocarditis, toxic; effect of digitalis, 1205
Simple pulse indicator (V. J. Keating), 1188
— standard exercise test and its use for measuring exertion dyspnoea (P. Hugh-Jones), 65 (O); 95 (A)
Sounds: *Herzschalldiagnostik in Klinik und Praxis* (J. Schmidt-Voigt), 1233
Surgery; valvotomy for mitral stenosis; further report, on 100 cases (C. Baker and others), 1043 (O); 1073 (L)
Tachycardia, paroxysmal, in pregnancy, 174

Heartburn, mechanism and treatment of, 1206
Heat, clinical effects of exposure to, 1345, 1402
— pyrexia and hyperpyrexia, 1345
HEATH, C.: Effect of reading on cataract, 651
Heating Appliances (Fireguards) Bill (parliamentary note), 665
HEATON, Nell, and DAYNES, G.: *Feeding Mothers and Babies*, 960
Heberden Society, 982
Heberden's nodes and generalized osteoarthritis (J. H. Kellgren and R. Moore), 181 (O); 208 (L)
Heel, wooden, for walking plasters, 710, 1300
HEFFERNAN, P.: Aluminium and silicosis, 1206
Height: Statures of a group of university students and of their parents (J. V. G. A. Durnin and J. B. de V. Weir), 1006 (O); (correspondence), 1133, 1300
Heliotherapy, 724
HELLIER, F. F.: Dermatologist's protest, 1406
Helminthiasis: Fits from African worms, 339
HEMINGWAY, A.: Fatigue, 815
HENDERSON, J. Y.: *Circus Doctor*, 1118
HENDERSON, N. P.: Shortage of *x*-ray films, 107, 385
HENDERSON, P.: School Health Service, 1022
HENDERSON, T. L. *See* DUNLOP, D. M., 292
HENLY, A. A. *See* COPEMAN, W. S. C., 397
HENRY, J. L.: Is Latin dead? 924
Heparin, prolonged anticoagulant therapy with (A. Wynn, J. F. Goodwin, and Angela Birbeck), 893 (O)
Hepatic duct: Repair of damaged accessory hepatic duct by anastomosis with cystic duct (H. Wapshaw), 906
Hepatitis, acute; treatment with vitamin K (H. Mossberg), 1382 (O)
— infective; a problem of world health (Sir John McNee), 1367 (O); 1399 (L)
— — intradermal test for, 1366
— — treated with A.C.T.H. and cortisone, 860
— toxic, during P.A.S. therapy (A. G. Fergusson, J. P. McIntyre, and A. R. Gemmell), 855
— viral; tattooing as possible means of transmission, 1111
HEPTINSTALL, R. H., and SOWRY, G. S. C.: Peripheral neuritis in systemic lupus erythematosus, 525 (O)
HERBERT, E. M.: Arterial occlusion during fit, 1303
HERD, D. A.: Medical aspects of tobacco smoking, 869
HERDAN, G. *See* HUDSON, G., 999
Hermaphroditism: Diagnosis of intersex (D. I. Williams), 1264 (O); 1292 (A)
Hernia, diaphragmatic, congenital in siblings (E. E. Philipp and M. O. Skelton), 1283 (O)
— epigastric, and associated dyspepsia, 220, 655
— inguinal, and prostatic obstruction; a new approach (T. Moore), 362; 547 (C), 605 (C)
Herpes gestationis, 230
— ophthalmicus, with subsequent Parkinson's syndrome (G. Strong), 533, (correspondence), 820
— treatment of post-herpetic neuralgia, 230
— zoster varicellosus (D. I. McCallum), 520 (O); (correspondence), 651
HERRON, R. A. C.: Inflation of lungs during bronchoscopy, 165
HERSHMAN, M.: Gastric carcinoma and acute perforation, 109
— Left-sided appendicitis, 1357
HERXHEIMER, H. G. J.: *Management of Bronchial Asthma: A Guide to Treatment*, 1394
HESELTINE, Michael, obituary of, 663, 716, 822
Hesperidin as oral contraceptive, 1183 (A)
Hetrazan treatment of onchocerciasis; histological study (F. Hawking), 992 (O)
HEWER, C. L.: Prolonged respiratory paralysis after succinylcholine, 971 (correction, 1084)
Hexamethonium. *See* Methonium compounds
HICKEY, M. D. *See* O'MEARA, R. A. Q., 605
HICKSON, Barbara. *See* GILLIES, H., 527
HIGGINS, T. T.: Centenary of "Great Ormond Street," 377; 371 (A)
— WILLIAMS, D. I., and NASH, D. F. E.: *The Urology of Childhood*, 695
HILL, A. B. *See* DANIELS, M., 1162
HILL, J. M. *See* HABERMAN, S., 851
HILL, Sir Leonard: Anoxia in sleep, 651
— obituary of, 767
HIMSWORTH, H. P., appointed K.C.B., 44
HIMWICH, H. E.: *Brain Metabolism and Cerebral Disorders*, 802
HINDE, R.: Tonsils and adenoids, 722
Hip: *Congenital Dislocation of the Hip* (J. Hass), 1287
— reconstruction by acrylic prosthesis, 48
Hirschsprung's disease; death following barium enema (A. Jolleys), 692 (O); (correspondence), 866
Hirsutism, idiopathic, steroid metabolism in, 39 (A)
Histamine liberation by anaphylatoxin, 781
— — in anaphylactic shock, 780
— mode of action of antihistaminic drugs, 380
— release as cause of hypersensitivity, 324
— skin test, value in Ménière's disease, 869
Histology: *Atlas of Histologic Diagnosis in Surgical Pathology* (K. T. Neubuerger), 748
History of medicine. *See* Medicine, history; Nova et Vetera
HOBBS, H. E.: Congestive and simple glaucoma, 869
HOBSON, A. C. S., FRASER, D. E., and NEWMAN, N. H.: Tattooing as possible means of transmitting viral hepatitis, 1111 (O)
HODGES, R. J. H.: Ryle's tube in trachea, 710
Hodgkin's disease, effect of triethylene melamine in, 1343 (A)
— — with thrombocytopenic purpura and amyloidosis (R. C. Gledhill and A. J. Shillitoe), 1337
— — lymph node regression as criterion of cure, 829
HODKINSON, R.: Intra-partum uterine rupture, 308; 438 (C), 604 (C), 973 (C)
HOLDSWORTH, W. G.: *Cleft Lip and Palate*, 695
Holland bequest to nationalized hospitals (medico-legal note), 1305
HOLLINS, C.: Emetine and scorpion stings, 614
HOLMES, Sir Gordon, awarded Conway Evans Prize, 797

Holmes, Sherlock: *My Dear Holmes. A Study in Sherlock* (G. Brend), 146
HOLORAN, Irene M.: Incidence and prognosis of cerebral palsy, 214
HOLZEL, A., KOMROWER, G. M., and WILSON, V. K.: Amino-aciduria in galactosaemia, 194 (O)
— *See* WILSON, Vera K., 358, 484
— *See* WOLMAN, B., 419
Honours Lists, 44 (correction, 118), 1299; 1293 (A)
HORACK, H. M. *See* SIMMONS, J. S., 1010
HORDER, Lord: Osler's fatal illness, 156
— dinner in honour of 81st birthday, 172
Hormone Assay (Editor: C. W. Emmens), 424
Hormone assays in normal and abnormal pregnancies (G. I. M. Swyer), 619 (O)
— therapy in endometriosis, 286
HOSFORD, J. *See* DUNLOP, E., 1392
Hospitality for overseas visitors, 1246

HOSPITALS:
Administrators' conference: Criticisms from the patient, 1244
Beds; Emergency Bed Service; applications and admissions, 56, 114, 228, 283, 338, 394, 446, 500, 555, 612, 668, 722, 776, 828, 877, 929, 983, 1039, 1090, 1142; tribute to, 1304
— emergency bureau at Liverpool, 283
— medico-legal aspect (medico-legal note), 496
— shortage, 322
Bequests to nationalized hospitals; complex problems (medico-legal note), 1200, 1251 (C); American legacy (medico-legal note), 1305
Connaught Military Hospital; conditions and accommodation (parliamentary note), 497
Cost, 760
Costs increased (parliamentary note), 773
Design of out-patient departments, 1022
Food costs per patient (parliamentary note), 497
Funds; vestment of Leith Hospital funds in Edinburgh Northern Hospitals Board, 926
Future growth in London, 555
General practitioner in hospitals, 151 (A); 1087 (parliamentary note)
Glasgow Royal Cancer Hospital changes name, 983
Greenwich and Deptford Hospital Management Committee annual report, 1384
Hospital for Sick Children, centenary of, 371 (A), 377, 1040, 1184 (A)
Hospital for Tropical Diseases, London, 1360
House appointments, purpose of, 914 (A)
International congress, 1187
Leith Hospital funds, 926 (medico-legal note)
Liverpool Heart Hospital, 1355
"Negligence" in hospital, 1341 (L)
Nottingham: *History of the General Hospital near Nottingham* by F. H. Jacob (V. Z. Cope) (Nova et Vetera), 272
Patients' feeding costs (parliamentary note), 1360
Prayers for Use in Hospitals, 534
Role of statistics in regional planning, with special reference to Birmingham Hospital Region (Vera Norris), 129 (O); (correspondence), 272, 437
Royal Beatson Memorial Hospital, 983
Royal Cancer Hospital; change of name suggested (parliamentary note), 1360
Royal Dental Hospital and London School of Dental Surgery, 1115
St. Thomas's Hospital; quatercentenary of refounding, 55
Staff, compulsory retirement of, 38 (A)
— future requirements, 322
Staffing cost (parliamentary note), 1200
Tiverton and District Hospital centenary, 1325
Unpunctual doctors (parliamentary note), 1412
Visiting children in hospital (A. Moncrieff and A. M. Walton), 43 (O); 824 (parliamentary note); for and against, 613; visiting hours for parents, 1286
Voluntary Service and the State. Study of the Needs of the Hospital Service, 367 (L); (correspondence), 657
West Middlesex Hospital League of Friends, 422
Westminster Hospital; Old Students' Dinner, 1309

HOUGHTON, R.: Epigastric hernia and dyspepsia, 220
HOULTON, A. C. L. *See* BEMBRIDGE, B. A., 675
House dust as an allergen, 35 (L)
HOUSEMAN, Joan: Family planning, 866
HOUSSAY, B. A., and others: *Human Physiology*, 585
HOWARD, A. V.: *Chambers's Dictionary of Scientists*, 310
HOWARD-JONES, John, obituary of, 978
HOWELL, T. H.: Stellate-ganglion block, 381
HUDSON, G., HERDAN, G., and YOFFEY, J. M.: Effect of repeated injections of A.C.T.H. upon the bone marrow, 999 (O); 1135 (C)
HUGHES, R.: Effect of sulphonamides on mortality, 972
HUGHES, T.: Death on the roads, 47
— Proctalgia fugax, 880
— Cowpox, 1249
HUGHES, W.: Cortisone in rheumatism, 601
HUGH-JONES, P.: Simple standard exercise test and its use for measuring exertion dyspnoea, 65 (O); 95 (A)
HUGO, Edward Victor, obituary of, 224
HUMPHREYS, J. *See* JELLIFFE, D. B., 405
Hungarian Microbiological Society; first congress, 331
Hungary: Kossuth Awards in medicine, 951
Hunger: *Geography of Hunger* (J. de Castro), 1234
— hypothalamic control of, 752 (A)
HUNT, E. F.: Doctor and the Courts, 50
HUNT, J. H.: College of General Practice, 1410
HUNTER, A. R.: Xylocaine, 714
HUNTER, D., and BIDSTRUP, P. L.: Poisonous chemicals in agriculture, 1135
— *See* BIDSTRUP, P. L., 277
Hunter, John, memorial tablet to, unveiled at St. Martin-in-the-Fields Church, 433
Hunter, William, memorial plaque at Lyric Theatre, 1020; unveiled, 1309
Hunter Medal and Triennial Prize, 875
Hunterian Oration, 56
Hunterian Society: Annual dinner, 1343, 1363
— — Essay competition, 1203
— — Gold Medal awarded to G. M. Temple, 1257
HURLEY, M. J., and MONRO, A. B.: Prolonged respiratory paralysis after succinylcholine, 1027
HURRY, C.: Fouling the nest, 974
Hydatidiform mole associated with full-time pregnancy, 1285
— — hormone levels in, 623
Hydrocortisone: Antirheumatic effects of hydrocortisone (free alcohol), hydrocortisone acetate, and cortisone (free alcohol) as compared with cortisone acetate; results from oral administration in patients with rheumatoid arthritis (E. W. Boland), 449 (O); 559 (L)
Hygiene: *Rosenau's Preventive Medicine and Hygiene*. 7th ed. (K. F. Maxcy), 1338
— *Translation of Galen's Hygiene* (*De Sanitate Tuenda*) by R. M. Green (Nova et Vetera) (C. Singer), 1404
L-Hyoscine hydrobromide as preventive against seasickness, 57, 1042
Hyperemesis gravidarum, hormone levels in, 623
Hypertension, management of (J. McMichael), 933 (O); 962 (L); 1083 (C), 1190 (C), 1191 (C), 1247 (C), 1248 (C)
— treatment, medical, 587 (L); 597; 709 (C)
— — with hexamethonium homologue (M. & B. 1863) (S. Locket and others), 254 (O)
— — with oral methonium compounds (A. J. M. Campbell, J. G. Graham, and R. D. H. Maxwell), 251 (O)
— — with salt-free diet, 1354
HYPHER, N. C.: Anaesthetic explosions, 273
Hypnosis: Damages against stage hypnotist (medico-legal note), 823
— in childbirth (A. M. Michael), 734 (O); (correspondence), 1132
— in medicine, 487
— in treatment of asthma, 173, 326, 487, 546, 816, 922
— on stage, film, and television, 326, 773, 816, 921
Hypnotics, quick-acting, 118, correction, 396
Hypnotism Bill (parliamentary note), 979
Hypochromasia, effect of, on specific gravity and sedimentation of red cells (K. B. Rogers), 1109 (O) (correction, 1418); 1407 (C)
Hypopituitarism; A.C.T.H. in treatment (C. F. Rolland and J. D. Matthews), 1220 (O)
— treatment of hypopituitary coma (H. L. Sheehan and V. K. Summers), 1214 (O)
— with pituitary tumours; cortisone in treatment of stupor associated with hypopituitarism (J. E. Caughey, A. James, and E. K. MacLeod), 1216 (O)
Hypoprothrombinaemia induced by dicoumarol and "tromexan," effect of vitamin-K preparations on (A. S. Douglas and A. Brown), 412 (O); 603 (C), 763 (C)
Hypotension, dangerous, after anaesthesia, 613
Hypothalamus in relation to hunger, 752 (A)
Hypothyroidism. *See* Thyroid
Hysteria treated with ethyl chloride, 1191

I

IBBOTSON, William, obituary of, 1359, 1411
Ice-cream, heat treatment of (vital statistics), 1037
Icterus gravis and cirrhosis of the liver (J. Gerrard), 1385 (O)
Identity cards abolished; numbers retained for National Health Service (parliamentary note), 497
— numbers for temporary residents (parliamentary note), 1412
Ileostomy in ulcerative colitis, 883
Illegitimate Child, Law in Relation to, 271; 265 (A)
Illustration, medical; exhibition at B.M.A. House, 1293, 1363
Immunity, artificial, 212
— in relation to use of chemotherapy, 1260
— natural, 212
Immunization: *Bacterial and Virus Diseases. Antisera, Toxoids, Vaccines, and Tuberculins in Prophylaxis and Treatment*, 2nd ed. (H. J. Parish), 91
— pros and cons (R. Cruickshank), 212, 268, 385, 714 (C)
— reactions, 213
Imperial Cancer Research Fund jubilee, 968
Imperial Chemical Industries Ltd., conspiracy action against (medico-legal note), 1139
INCH, R. S. *See* DUNLOP, D. M., 292
Income tax and Spens, 913 (A)
Incontinence. *See* Urine, incontinence
Incontinentia pigmenti; report of case, with summary of 29 cases from the literature (A. W. Franklin), 75 (O); (correspondence), 277, 439
Index Bibliographicus, 1090
India: Population (1951), 1224
— Sickle-cell trait (H. Lehmann and Marie Cutbush), 404 (O); 426 (L)
INDUSTRIAL MEDICINE: *See also* Mining; Toxicology
Accidents and diseases (vital statistics), 54, 337, 553, 827, 981, 1256
Dangers from penetrating oils, 829
Health of industrial workers, 1023
Industrial Health Research Board reports, 393
Injuries (parliamentary notes), 391
Tuberculosis in industry, 1012 (L)
Welding and health, 1239 (A)

INFANTS: *See also* Children
Adaptability to changes of climate, 286
Antibiotics for underweight infants, 985
Appendicitis, clinical picture, 777
Bladder control, 669
Breast-feeding and tuberculosis, 879
Cortical hyperostosis (W. J. Matheson and Mary Markham), 742 (O)
Diabetes mellitus, two cases in infants under 1 year (H. S. Brodribb, J. McMurray, and L. G. Scott), 1060 (O); 1354 (C)
Feeding Mothers and Babies (Nell Heaton and G. Daynes), 960
Kernicterus without prematurity or blood-group incompatibility (N. R. Butler and W. G. Spector), 1168 (O)
Laryngoscope blade for infants (R. Bryce-Smith), 217
Megaloblastic anaemia of infancy (A. G. Rickards), 1226 (O)
Mortality rate (vital statistics), 169, 982
— — (vital statistics), 282
Newborn, acute suppurative parotitis in (R. H. R. White), 1232
— care of; instruction of the medical undergraduate (W. S. Craig), 1062
— *Foetal and Neonatal Pathology* (J. E. Morison), 639
— haemolytic disease of, 108
— mucus extractor for (H. H. F. Barns), 598
— poliomyelitis in mother and infant (G. A. Wright and T. K. Owen), 800 (O)
— priapism in, 614
— respiration, 97 (A)
— urticaria in, 1134
Perversion of appetite; cause and treatment, 778
Study on the A factor as a cause of haemolytic disease in premature babies (S. Phansomboon and O. J. Pollak), 1170 (O)

Infection, air-borne, prevention of, 1259
— cross-infection among children (parliamentary note), 1412
— spread in the family, 968
— sterilization of hospital blankets (H. F. Barnard), 21 (O); 601 (C)

INFECTIOUS DISEASES:
Graphs, 54, 114, 170, 226, 282, 336, 392, 445, 498, 553, 610, 666, 721, 774, 775, 827, 876, 928, 981, 1038, 1088, 1140, 1202, 1256, 1308, 1362, 1363, 1414–5
Incidence in 1951 (vital statistics), 169
Notification of disease, 474 (L); 713 (C)
Notifications (vital statistics), 54, 113, 169, 226, 282, 336, 392, 444, 498, 553, 610, 666, 720, 775, 826, 876, 928, 981, 1037, 1089, 1140, 1202, 1256, 1308, 1362, 1414
Weekly table, 55, 113, 171, 226, 281, 337, 391, 444, 498, 554, 611, 667, 720, 774, 826, 877, 927, 980, 1037, 1088, 1140, 1202, 1257, 1308, 1362, 1414

Infertility. *See* Fertility and infertility
Inflammation, 1123 (A)
Influenza, epidemiology, 209 (A)
— gastric, differential diagnosis, 396
— Glasgow outbreak (vital statistics), 720
— graphs (vital statistics), 171
— immunization, 38 (A); 270
— incidence and type distribution of capsulated *H. influenzae* strains (Betty Dawson and K. Zinnemann), 740 (O)
Injuries, personal; assessment of damages (medico-legal note), 605
INMAN, P., and COWAN, I. C.: Antihistamines in non-urticarial dermatoses, and a retrial of antihistamine iontophoresis, 1064 (O)
Insanity and criminal responsibility, 45; 50 (C)
— certification of, 754
— incurable, as grounds for divorce (medico-legal note), 52
Insecticides: Agriculture (Poisonous Substances) Bill (parliamentary notes), 1035, 1199, 1252, 1360
— poisoning due to; blood cholinesterase level in diagnosis (W. N. Aldridge and D. R. Davies), 945 (O); 1015 (L); (correspondence), 1135
— — — — laboratory tests for agricultural workers, 1014 (A); 1024
— symposium at Royal Society of Tropical Medicine and Hygiene, 543
— toxic effects of *bis*-mono-*iso*propyl aminofluorophosphine oxide, 277
— toxicity to man, 544
Insemination, artificial (donor); procedure and legal aspects, 1259
Institute of National Administration: *The Health Services; a Compendium of Conference Papers*, 803
Insulin: Duration of action of different insulins (F. Gerritzen), 249 (O); 440 (C)
— hypertension as cause of "fits," 395
— hypoglycaemia treatment of gout, 1251
— shock therapy, arterial occlusion during, 1303
— — — dosage in schizophrenia, 501

Insurance and pensions, 207 (L)
— statistics for study of sickness, 753 (A)
— *see also* National Insurance
International Association of Gerontology and rapid progress of gerontology (V. Korenchevsky), 375
International exchange of medical facilities (parliamentary notes), 1035
International Fertility Association, 1203
International Tuberculosis Campaign: Mass B.C.G. vaccination campaign in Malta (V. Zammit-Tabona), 837 (O)
Intervertebral disk lesions; conservative treatment, 217
— — — hazards in manipulative treatment of lumbo-sacral protrusions (R. Hafner), 361 (O); (correspondence), 655
— — — sponge-rubber collar for, 972
Intestines: Meckel's diverticulum, bleeding; report of two cases (J. N. Walton and Norma D. Lill), 88 (O); correspondence, 221
— obstruction, duodenal, congenital, with mongolism (M. Bodian and others), 77 (O); (correspondence), 276, 384
— small; extensive resection; short review, with two new cases (H. J. Croot), 195 (O); (correspondence), 654
— stricture and anastomosis associated with macrocytic anaemia (G. M. Watson and L. J. Witts), 13 (O)
Intracranial haemorrhage, spontaneous, in the young, 1013 (L)
IOVETZ-TERESHCHENKO, N. N.: Absence of appendix, 1302
IRELAND, J. T. *See* COHEN, B. E., 48
Ireland, Republic of
Dublin 1660–1860. *A Social and Architectural History* (M. Craig), 1395
Posts in British hospitals for newly-qualified doctors, 847
Registrar-general's quarterly return, 226, 1256
Report of Department of Health for 1950–1 (vital statistics), 1255
Iridocyclitis treated with streptomycin, 121
Iritis, recurrent; causes and treatment, 1366
— treatment of, 318
Iron therapy; effect on "physiological" anaemia of pregnancy (Nancy Benstead and G. W. Theobald), 407 (O); 476 (A); 599 (C), 817 (C)
— — in anaemia, 599
IRVINE, E. D.: Notification of disease, 713
IRWIN, D. B.: Septic jaundice; a complication of peritonitis following gastro-intestinal surgery, 1379 (O)
Isle of Man: Rising costs of Health Service (parliamentary note), 927
ISMET-ANWAY, M.: Gastro-duodenal perforations, 330
*Iso*nicotinic acid hydrazide in treatment of tuberculosis, 478, 858 (L), 983, 1015 (A); (correspondence), 868, 970, 1028, 1081, 1133; Ministry of Health statement, 775
1-*Iso*nicotinyl-2-isopropyl hydrazine, 858
Isotopes in medicine, 102
— radioactive; use at University College Hospital, 648, 667
Israel: Rabies incidence, 244
Italy; study tour in 1952, 338
IVES, J.: Forgotten tourniquet, 328

J

JACKSON, C. R. S. *See* BEMBRIDGE, B. A., 675
JACKSON, D. M. *See* BULL, J. P., 1018
JACKSON, D. S. *See* KELLGREN, J. H., 997
JACKSON, Henry Law, obituary of, 1360
JACKSON, J. N.: Death under anaesthesia, 715
JACKSON, W. P. U.: Extensive resection of the small intestine, 654
Jacksonian Prize, 875
JACOB, F. H.: *History of the General Hospital near Nottingham* (V. Z. Cope) (Nova et Vetera), 272
JACOBI, J.: *The Psychology of C. G. Jung*, 5th ed., new and revised, 310
JACOBS, P.: Attempted suicide, 490
JAMES, A. *See* CAUGHEY, J. E., 1216
JAMES, C. D. T.: Ryle's tube in the trachea, 715
James D. Bruce Memorial Lectureship and Medal awarded to J. H. S. Gear, 1090
JAMES, Irene M.: Family planning, 1247
JAMES, T.: Pink disease and infections, 709
JAMIESON, S. R.: Tuberculous meningitis, treatment with streptomycin and *para*-aminosalicylic acid, 83 (O), (corrections, 165, 223), 329 (C)
JANUS, O. *See* KELLGREN, J. H., 997
JARVIE, H. F.: Subconvulsive dosage in shock therapy, 1082
Jaundice, homologous serum; survey of patients receiving irradiated plasma, 347
— icterus gravis and cirrhosis of liver (J. Gerrard), 1385
— relations of homologous serum jaundice and syringe-transmitted jaundice to acute infective hepatitis, 1368
— septic; a complication of peritonitis following gastro-intestinal surgery (D. B. Irwin), 1379 (O)
JELLIFFE, D. B., and HUMPHREYS, J.: Sickle-cell trait in Western Nigeria: Survey of 1,881 cases in the Yoruba, 405 (O); 426 (L); (correspondence), 763, 920
JENNINGS, G. H.: Clinical features of the pneumonias undergoing virus tests, 123 (O)
JENNINGS, P. B.: Adrenal cortical function at death as measured by level of circulating eosinophils, 1055 (O); 1249 (C)
JESSOP, W. J. E. *See* O'MEARA, R. A. Q., 605
Jocasta complex in adoptive mothers, 817
JOHNSON, D. H. *See* GRAYSON, J., 546
JOHNSTON, J. M.: Ethics of leucotomy, 1131
JOHNSTON, L.: Medical aspects of tobacco smoking, 869
Joint Tuberculosis Council: Joint annual report, 7; annual luncheon, 1203
Joints, ankylosis due to myositis ossificans progressiva, 954
JOLLEYS, A.: Death following a barium enema in a child with Hirschsprung's disease, 692 (O)
JONES, B. F. S.: Family planning, 1028
JONES, E.: *Essays in Applied Psycho-analysis*, vol. 2, 261
— Penicillin and colds, 385
JONES, E. S.: Medical aspects of tobacco smoking, 869
JONES, F. W.: Soviet genetics, 708
JONES, G. B.: Intra-articular streptomycin, 766
JONES, I. C.: What doctors prescribe, 486
JONES, O. G. *See* WALLACE, E. J. G., 1231
JOSEPHS, S. *See* CAMPBELL, H., 1408
JOURNALS:
Abstracts of World Medicine, publication changes, 1204
Abstracts of World Surgery, Obstetrics, and Gynaecology, incorporated in *Abstracts of World Medicine*, 1204
American Journal of Tropical Medicine, 55
American Orthoptic Journal, 172
Annals of Physical Medicine, 445
Applied Statistics, 1089
Australasian Annals of Medicine, 115
Experimental Parasitology, 172
Family Doctor, 39 (A)
Great Ormond Street Journal, 392
Journal of the National Malaria Society, change of title, 55
Journals for Yugoslavia, 1030
Laboratory Investigation, 338
Medical and Biological Illustration, 98 (A); 1293 (A)
Publication difficulties of scientific and medical journals, 283
Review of Medical and Veterinary Mycology, 1400 (A)
University of Durham Medical Gazette, 1257
University of Leeds Medical Journal, 611
West African Medical Journal, 877
West Indian Medical Journal, 152
Your Health, 1203
JOYNER, Joan. *See* MELLANBY, Lady May, 7
Judo neck-strangle, 447
JUDSON, D. H.: Is metallic mercury poisonous? 1406
Jung: A great thinker, 314 (L); 655 (C)
— *The Psychology of C. G. Jung*, 5th ed., new and revised (J. Jacobi), 310
Jurisprudence, medical. *See* Medico-legal

K

Kahn tube test with dropping-pipettes (O. Khairat), 582 (O)
Kala-azar; discovery of the formol gel reaction, 163
"Kapilon," effect in hypoprothrombinaemia, 414
KAREL, L., and ROACH, Elizabeth S.: *A Dictionary of Antibiosis*, 1290
KATZ, B., elected F.R.S., 703
KAUNTZE, R.: Hypotensive effects of "veriloid," 598
KEATING, V. J.: Simple pulse indicator, 1188
KEAY, K. R. *See* THOMSON, J., 295
KEELE, K. D.: *Leonardo da Vinci on Movement of the Heart and Blood*, 802
KEIDAN, S., LYONS, J., MANN, B., and WILTHEW, G. A.: Tuberculosis survey in school-children in the Calder Valley, 1390 (O); 1397 (L)
KEIDAN, S. E. *See* COHEN, B. E., 48
KELLEY, Marion. *See* MELLANBY, Lady May, 7
KELLGREN, J. H.: Some concepts of rheumatic disease, 1093 (O), 1152 (O)
— JANUS, O., MOORE, R., and JACKSON, D. S.: Responsiveness to A.C.T.H. and cortisone in rheumatoid arthritis, 997 (O); 1016 (A) (correction, 1144)
— and MOORE, R.: Generalized osteoarthritis and Heberden's nodes, 181 (O); 208 (L); 383 (C), 765 (C)
KELLIE, A. E. *See* COPEMAN, W. S. C., 397
KEMP, L. A. W.: *Students' Radiological Mathematics: Elementary Mathematics Applied to Medical Radiology*, 908
KENNEDY, A.: Psychiatric case and the law: 1. General principles, 754; 2. Admission to hospital, 810; (correspondence), 921, 971, 1081, 1082, 1193, 1303, 1407
KENNEDY, Robert Foster, obituary of, 165, 223
KENNEFICK, M. J.: Mental disease a symptom of physical defect, 972
KENNY, Elizabeth: *And They Shall Walk. The Autobiography of Sister Elizabeth Kenny*, 802; 1082 (C)
Keratitis, tuberculous, treated with streptomycin, 121
Keratoderma climactericum, 1206
Kernicterus without prematurity or blood-group incompatibility (N. R. Butler and W. G. Spector), 1168 (O)
KERSLAKE, D. McK. *See* COOPER, K. E., 1409
KERSLEY, G. D., WATSON, D., and MANDEL, L.: Splenins A and B in rheumatoid arthritis, 438, 603
KESSEL, A. W. L.: Invite Russian doctors, 50
KESSEL, I.: Combination of congenital anomalies, 470; 762 (C), 1249 (C)
17-Ketosteroid excretion in idiopathic hirsutism, 39 (A)
KEYNES, G. (Editor): *Apologie and Treatise of Ambroise Paré* (C. Singer) (Nova et Vetera), 1129
KHAIRAT, O.: Kahn tube test with dropping-pipettes, 582 (O)
Kidney: Mercury nephrosis in young children, with special reference to teething powders containing mercury (Vera K. Wilson, M. L. Thomson, and A. Holzel), 358 (O); 370 (A); (correspondence), 484, 931
— structural and functional adaptation in renal failure (R. Platt), 1313 (O), 1372 (O); 1396 (L)
— tuberculosis, bilateral, treatment of, 396
— *Kidney: Medical and Surgical Diseases* (A. C. Allen), 423
KILOH, L. G., and NEVIN, S.: Isolated neuritis of anterior interosseous nerve, 850 (O)
KILROE, Laurence, obituary of, 1197, 1252
KING, A. C.: Trichlorethylene analgesia in labour, 328
KING, J. J. D.: Appeal for specimens, 549
King Edward's Hospital Fund for London: *Voluntary Service and the State. Study of the Needs of the Hospital Service*, 367 (L); (correspondence), 657
King George VI, death of, 366 (L); 386 (correction, 549)
King's College Hospital Medical School: Wiltshire Memorial Research Scholarship, 1307
KINGSTON, William George, obituary of, 1198
KINMONTH, J. B.: Physiology and relief of traumatic arterial spasm, 59 (O); 93 (L)
— and HADFIELD, G. J.: Sympathectomy for Raynaud's disease; results of ganglionectomy and pre-ganglionic section compared, 1377 (O)
KINSELLA, V. J.: Johann Mikulicz-Radecki, 975
KIRKPATRICK, H. F. W. *See* ROBERTSON, J. D., 624
KITCHING, R. L.: Experiment in peacemaking, 548
KLEIN, G. *See* PECK, S. M., 1011
KLEITMAN, N.: Physiology of sleep, 971
KNAYSI, G.: *Elements of Bacterial Cytology*, 2nd ed., 747
Knee-joint, tuberculosis of; treatment with intra-articular streptomycin (R. T. Ahern and G. P. Arden), 466 (O); (correspondence), 766, 974, 1194
KNOWLES, G. S. A.: Modified endotracheal connector, 1355
KNOX, R.: Drug resistance of micro-organisms, 434
— MACLEAN, K. S., and ROBSON, J. M.: New results with *iso*nicotinic acid hydrazide, 1081
Koch phenomenon in mass B.C.G. vaccination, 839
Kokka disease, 477
KOMROWER, G. M. *See* HOLZEL, A., 194
KONDI, A. *See* FOY, H., 852
Korea: Bacteriological warfare, 652
— epidemic haemorrhagic disease, 477 (A)
— Manchurian fever, 352
— 38th Parallel Medical Society of Korea, 1415
KORENCHEVSKY, V.: The International Association of Gerontology and rapid progress of gerontology, 375
Korin fever, 477
KREMER, M.: Conservative treatment of disk syndrome, 217
Kroll's treatment of brain injuries (parliamentary note), 609

L

Labia minora, periodic oedema of, 1092
Laboratory Investigation, 338
LABOUR: *See also* Abortion; Caesarean section; Infant, newborn; Obstetrics; Pregnancy; Puerperium
Analgesia with trichlorethylene, 108
Disproportion; accuracy of *x*-ray cephalometry *in utero*, 1026
Domiciliary midwifery, 1135, 1246, 1352
Hypnosis in childbirth (A. M. Michael), 734 (O); (correspondence), 1132
Intra-partum uterine haemorrhage (R. Hodkinson), 308; 438 (C), 604 (C), 973 (C)
Preparation for "natural childbirth," 1302
Present conception of trial labour, 1025
LACE, Frederick, obituary of, 1198
Lacrimal glands, involvement in mumps, 1069; 1250 (C)
Lactation, arrested, fate of milk after, 985
— physiology of, 1340 (L)
Lady Tata Memorial Trust; grants, scholarships and fellowships, 114, 1363
Laevulose, sobering effects of, 1041
Lagos Eye Clinic, 584
LAING, G. D.: Test for anaemia, 600
LAMBERT, A. V. *See* HUGH-JONES, P., 65
LAMBIE, R. S.: Experiment in peacemaking, 652
Lambliasis associated with acute infectious lymphocytosis, 79
LANDER, O. LL.: Trichlorethylene analgesia in labour, 108

Langdon-Brown, Sir Walter (G. Evans), 175 (O); 206 (L)
Langdon-Brown Lecture (G. Evans), 175, 206
Language disabilities, 36 (A); 325
LARSELL, O.: *Anatomy of the Nervous System*, 2nd ed., 1010
Laryngeal epilepsy, 653, 1250
Laryngoscope blade for infants (R. Bryce-Smith), 217
Larynx: Aid to analgesia of the larynx and trachea (T. Dinsdale) (preparations and appliances), 707
— paralaryngeal actinomycosis in a child (D. F. Davies), 363
LASK, A.: Discharge of certified mental patients, 1193
Latin, is it dead? 924, 1084, 1197, 1304
Latin America, medical mission to (T. M. Ling and others), 756
LAUFER, W. E.: Some observations on subtertian malaria, 1281 (O)
LAURENT, L. J. M., and PARISH, H. J.: Serum reactions and serum sensitivity tests, 1294
LAURIE, J. H.: Skin resistance to ultra-violet light, 162
LAURIE, Jean: Family planning, 868
LAWLER, L. J. *See* LAWLER, Sylvia D., 856
LAWLER, Sylvia D., and LAWLER, L. J.: *Human Blood Groups and Inheritance*, 856
Laxatives, mild, for the elderly, 502, 830
LEACOCK, A.: Appendicitis in the West Indies, 1347
Lead as food contaminant, 218
— poisoning (parliamentary note), 1087
— — Effect of water softeners on lead pipes, 1417
— workers, surveillance of, 931
LEADING ARTICLES:
Abortion, habitual, 427
A.C.T.H. and cortisone in infective hepatitis, 860
Antibiotic treatment, superinfections during, 537
— What is an antibiotic? 1290
Award and the Working Party, 1235
Bacterial resistance to chemotherapy, 425; 546 (C), 712 (C)
B.C.G. vaccination, efficacy of, 147
Bevan's book, 749, (correspondence), 870, 974, 1030
Blood vessels: Vascular spasm, 93; 382 (C)
Bottle of medicine, 149; 326 (C)
Cancer: Progress of cancer, 1119; 1304 (C)
Carcinoma of stomach, 313
Certification, medical, of cause of death, 641
Colitis, ulcerative, surgery of, 910; 1194 (C)
Coronary disease in doctors, 535, 651
Cortisone and hydrocortisone, 588
— in carditis, 1179
Deafness: diagnosis in children, 264
Demographic revolution, 1289
Economics and doctors, 642; 818 (C)
Fatigue, 805
Forty million pounds, 697
Growth promoters, 640; 1357 (C)
Hepatitis, virus, epidemiology of, 1399
House dust as an allergen, 35
Hypertension, epidemiology of, 962; 1083 (C), 1190 (C)
— medical treatment of 587; 709 (C)
Insurance and pensions 207
Intracranial haemorrhage, spontaneous, in the young, 1013
Jung: A great thinker, 314; 655 (C)
King George VI, death of, 366, 386 (correction, 549)
Leonardo da Vinci, 804
Leucotomy, ethics of, 909; 1028 (C), 1131 (C), 1192 (C), 1250 (C)
Lung resection for pulmonary tuberculosis, 34
Marriage, divorce, and the B.M.A., 1237
Men and mountains, 1120
Mental Health Research Fund, 699; 975 (C)
Milk, secretion, 1340
Money and medicine, 749
N.H.S.: Charges proposed, 312
— Estimates, 701
"Negligence" in hospital, 1341
Notification of disease, 474; 713 (C)
Oesophagus, surgery of, 961; 1250 (C), 1358 (C)
Opticians, future for, 1074
Osteoarthritis, 208
Our Patron the Queen, 1340
Place for voluntary work, 367; 657 (C)
Plastics in surgery, 750
Preparing the case, 263
Queen Elizabeth II, accession of, 367
Radiology: Shortage of *x*-ray films, 94, (correspondence), 107, 219, 274, 327, 600
Renal failure, 1396
Retrolental fibroplasia, 698
Secret remedy for cancer, 1236
Sherrington, Sir Charles S., 589
Sickle-cell trait, 426
Sleep, physiology of, 745
Sympathectomy for cerebral ischaemia, 262; correspondence, 382, 487
Thus we are men, 206
Tuberculosis, chemotherapy of, 858, 1172
— in industry, 1012
Tuberculous infection, 1397
Valvotomy for mitral stenosis, 1073
LEAK, W. N.: What doctors prescribe, 484
— Social trends and home confinements, 1135, 1352
LEAROYD, C. G.: Criminal responsibility, 50
LEDERMANN, E. K.: Nature cure, 764
LEDGARD, Herbert Arthur, obituary of, 1033
LEE, B.: Is illness inevitable?, 1196
LEE, J. A.: Stellate-ganglion block, 381
Leeches: dimethyl phthalate as leech repellent, 898
Leeds University. *See* Universities: Leeds
LEES, A. W.: Acute polyarthritis with pulmonary consolidation and pleural effusion, 246 (O); (correspondence), 765
LEESE, W. L. B.: Spontaneous arterial occlusion during an epileptic fit, 854; 1303 (C)
Left hand, right hand, 591 (A); 763 (C)
Leg, fracture; heel, wooden, for walking plasters, 710, 1300
— progressive bacterial synergic gangrene of (R. Marcus), 1230
LEGGAT, P. O., and GIFFORD, J. H.: Uncommon neurotoxic symptom associated with dihydrostreptomycin therapy, 1008; 1302 (C)
LEHMANN, F. M.: Nature cure, 653
— Theory of cancer treatment, 1301
LEHMANN, H., and CUTBUSH, Marie: Sickle-cell trait in Southern India, 404 (O); 426 (L); (correspondence), 920
LEISHMAN, A. W. D.: Management of hypertension, 1247
— *See* SNEDDON, I. B., 1276
Leith Hospital funds (medico-legal note), 926
LEITNER, Z. A.: The nutritional revolution, 384
— Anaemia in pregnancy, 817
LEJEUNE, F.: *German-English Dictionary for Physicians*, 1118
LEMBERG, R., elected F.R.S., 703
LEMPERT, H.: Treatment of hypoprothrombinaemic haemorrhage, 603
LENNON, G. G.: Addison's disease and pregnancy, 708
— Stress incontinence; a preliminary report on new method of treatment, 1384 (O)
Lens, acrylic, intra-ocular, 1121 (A)
— Retrolental fibroplasia; problem of prematurity (B. A. Bembridge and others), 675 (O); 698 (L); (correspondence), 924, 1029
Leonardo da Vinci, quincentenary of birth of, 804 (L); Italian celebrations at Florence, 1142
— *Leonardo da Vinci on Movement of the Heart and Blood* (K. D. Keele), 802
Leprosy: Use of A.C.T.H. and cortisone, 601, 602
LERCHE, W.: *The Esophagus and Pharynx in Action. A Study of Structure in Relation to Function*, 639
LERICHE, R.: Treatment of embolism and thrombosis of the cerebral vessels, 231 (O); 262 (L); (correspondence), 381, 486, 487, 777
LESLIE, Leonard, obituary of, 872
Leucorrhoea, post-natal, 348
Leucotaxine, 1123 (A)
Leucotomy, ethics of, 909 (L); (correspondence), 1028, 1131, 1192, 1250
— follow-up study of 238 patients (H. Gillies, Barbara Hickson, and W. Mayer-Gross), 527 (O); 909 (L)
— *Frontal Lobotomy and Affective Behaviour: A Neurophysiological Analysis* (J. Fulton), 1394
— prefrontal; views of Scottish Analytic Group, 919
— undesirable effects, 49
LEUKAEMIA:
Anticoagulant treatment of thromboses, 162
Bullous lesions in (R. Scutt), 139 (O)
Effect of A.C.T.H. and cortisone, 1262
Grants, scholarships, and fellowships for research, 114, 1363
Granulocytic, splenogram in, 988
Lymphocytic, splenogram in, 988
Spontaneous rupture of spleen in (G. H. Cooray), 693
With primary gynaecological manifestations (C. E. W. Wheaton), 30
Leukonychia, 985
LEVENE, L. J.: Night pain with fractured clavicle, 1408
Leverhulme research grants and scholarships, 875, 1036, 1415
LEVERTON, J. S.: Case of interstitial tubal pregnancy, 421
LEVIN, B. *See* GORDON, I., 463
LEVIN, Joseph John, obituary of, 1085
LEWIN, B. D.: *Psychoanalysis of Elation*, 364
LEWIS, C. N. *See* WELCH, H., 1339
LEWIS, G. W.: Antihistamine drugs and skin eruptions, 1405
LEWIS, P. C.: Initiative, 223
LEWIS, R.: Professional prospects; comparison of the position of doctors with that of other professional people, 615 (O)
LEYS, D., and CAMERON, K.: Psychiatric study of 6 cases of infantile acrodynia, 191 (O)
LEYTON, N.: Migraine, 107
Libel and slander: Defamation (Amendment) Bill, 266 (A)
Liberia: Tropical medicine institute, 393
Libido, diminution in over-sexed girls by hormone administration, 986
LIBRACH, I. M.: Case of glandular fever with cerebral complications, 956 (O)
Libraries: Help in establishing new libraries and information services, 227
Library Association; Medical Section, weekend conference, Liverpool, 87, 813
LIDDIATT, J. E.: Foreign body in the eye, 975
LIDWELL, O. M.: Common cold, 969
Lighting, fluorescent, position in drawing-office, 230
LILL, Norma D. *See* WALTON, J. N., 88
Limbs, artificial; gadgets for the armless, 230, 778
LIMERICK, Earl of, appointed chairman of Medical Research Council, 227
LINDARS, D. C. *See* GLENNIE, J. S., 1009
LINDENBAUM, K.: Goat's milk and infantile eczema, 973
LING, T. M., BACH, F., SELLORS, T. H., and BROWN, A. I. P.: Mission to Latin America, 756
Lip: *Cleft Lip and Palate* (W. G. Holdsworth), 695
LIPMAN, B. S., and MASSIE, E.: *Clinical Unipolar Electrocardiography*, 1287
Lipoma of hypopharynx (J. B. Penfold), 1286
LIPP, G. R.: Diabetes in infancy, 1354
Liquor sulphestolis for domestic dish-washing, 724
Liquorice as cause of fatal hypokalaemic alkalosis with tetany during P.A.S. therapy (N. J. Roussak), 360 (O)
Lister Oration, 877
Listerian Festival, 877
LISTON, R. P.: Cricket, 1251
LITTLER, T. R.: Polyarthritis with pulmonary signs, 765
— and ELLIS, G. R.: Gall-stones; a clinical survey, 842 (O); 1031 (C)
Livedo reticularis, treatment of, 396
LIVER: *See also* Hepatitis; Jaundice
Cirrhosis and icterus gravis (J. Gerrard), 1385 (O)
— splenogram in, 989
Effect of liver supplement on growth of children (J. Yudkin), 1388 (O); 1401 (A)
Liver Disease (Editor: Sheila Sherlock), 1233
Repair of damaged accessory hepatic duct by anastomosis with cystic duct (H. Wapshaw), 906
Risks of needle biopsy (R. Terry), 1102 (O); 1249 (C)
Liverpool: Emergency bed bureau, 283
— Heart Hospital, 1355
— Medical Institution: Some problems in dermatology, 555
— University. *See* Universities: Liverpool
LIVINGSTONE, J. L. *See* SELLORS, T. H., 747
Lloyd, Dame Hilda, bronze bust unveiled, 983
LLOYD, J. P. F.: Congestive and simple glaucoma, 712
Lobotomy. *See* Leucotomy
LOCKET, S., SWANN, P. G., GRIEVE, W. S. M., and PLAYER, H. P.: M. & B. 1863, a homologue of hexamethonium, in treatment of hypertension, 254 (O)
LOCKHART, R. D.: Growth and development of children, 1133
LODGE, E.: Lysivane in parkinsonism, 490
LODGE, T. *See* COLVER, T., 89
LOGAN, W. P. D.: Nomenclature of diseases and operations, 493
— and MORRIS, J. N.: Doctors and heart disease research, 1190
LOGIE, John William, obituary of, 550
London; health in 1951 (vital statistics), 113
— water supply in wartime, 539 (A)
London County Council, J. A. Scott appointed Medical Officer of Health and School Medical Officer, 446
London Hospital Medical College, C. F. Barwell appointed Professor of Bacteriology, 1361
London School of Hygiene and Tropical Medicine, survey of work during 1950–1, 1039
London University. *See* Universities: London
LONG, D. A. *See* HART, P. D., 680
LOUW, J. H. *See* BODIAN, M., 77
LOVE, E. B.: Tablet test for urinary chloride, 275
LOVE, McN.: Pulmonary embolism, 920
LOVE, W.: Intra-partum uterine rupture, 604
LOWE, Godfrey John Ralph, obituary of, 1032
LOWE, J.: A.C.T.H. and cortisone in leprosy, 601
LOWENFELD, Margaret: Specific language disability, 325
LOXTON, G. E.: Splenins A and B in rheumatoid arthritis, 603
LUCE, Sir Richard Harman, obituary of, 493, 662
Lumleian Lectures, 1313
LUMSDEN, E. G. S.: Isonicotinic hydrazide, 1028
LUNGS:
Aspergillosis following antibiotic treatment of post-influenzal broncho-pneumonia (J. D. Abbott and others), 523 (O); 537 (L)
Carcinoma; relation to tobacco smoking, 335
Consolidation and pleural effusion associated with acute polyarthritis (A. W. Lees), 246 (O); (correspondence), 765
Death after 19 years from tooth in lung (medico-legal note), 718
Inflammation; accumulation of diethylaminoethanol ester of penicillin in inflamed lung tissue (J. Ungar and P. W. Muggleton), 1211 (O); 1238 (A)
Inflation during bronchoscopy, 165
Lung Abscess (R. C. Brock), 1287
Pneumonectomy; first successful operation, 986
— for bronchiectasis; indications and contra-indications, 557
Resection for pulmonary tuberculosis, 34 (L)
Sarcoma, secondary, causing spontaneous pneumothorax, 713
Simple standard exercise test and its use for measuring exertion dyspnoea (P. Hugh-Jones), 65 (O); 95 (A)
Lupus erythematosus, complications following mepacrine therapy, 1406
— — discoid, treatment and prognosis, 117
— — systemic, with peripheral neuritis (R. H. Heptinstall and G. S. C. Sowry), 525 (O)
LUTWYCHE, V. U., and BREWERTON, D. A.: Bone marrow depressed by thiosemicarbazone, 1302

LYDON, F. L.: Critique of therapy in rheumatoid arthritis, 602
Lymph glands, tuberculous, treatment of, 285, 830
Lymphocytosis, infectious, acute; report on cases in day nursery (H. G. Dunn), 78 (O); 96 (A); 324 (C)
Lymphosarcoma, effect of triethylene melamine in, 1343 (A)
LYONS, J. *See* KEIDAN, S., 1390
— splenogram in, 988
"Lysthenon." *See* Succinylcholine chloride

M

M. & B. 1863. *See* Methonium compounds
M.5943 therapy in malaria, 571
MACBETH, Archibald, obituary of, 332
MCCALLUM, D. I.: Herpes zoster varicellosus, 520 (O); (correspondence), 651
MCCAREY, A. G.: Balantidiasis in South Persia, 629 (O); 1357 (C)
MCCONNELL, A. A.: Pneumoencephalography and fits, 1194
MACDONALD, D. D.: What doctors prescribe, 599
MACDONALD, G.: Residual insecticides, 543
MACDONALD, I.: Growth of biparietal diameter of foetal head in last weeks of pregnancy, 798 (O); (correspondence), 922, 1408
MACEWAN, Archibald, obituary of, 550
MCFADZEAN, J. A.: Investigations into the cause of microfilarial periodicity, 1106 (O)
MCFARLAN, A. M. *See* BARRETT, A. M., 1317
MCFARLAND, J.: Educating the public about cancer, 656
MCFIE, J.: Invite Russian doctors, 50
MCGIBBON, John Paton, obituary of, 1031
MCGILL, D.: What doctors prescribe, 709
MCGREGOR, I. A., and SMITH, D. A.: "Daraprim" in treatment of malaria; study of its effects in falciparum and quartan infections in West Africa, 730 (O)
MCINTYRE, J. P. *See* FERGUSSON, A. G., 855
MACKENNA, R. B.: Some problems in dermatology, 555
MCKENZIE, W.: *Ear, Nose, and Throat Diseases for the General Practitioner*, 473
Mackenzie Industrial Health Lecture, 1364
MCKEOWN, T., and GIBSON, J. R.: Period of gestation, 938 (O); (correspondence), 1132, 1192, 1354
MACKEY, H. O.: *Handbook of Diseases of the Skin*, 1071
MACKIE, Alexander Hendry, obituary of, 1033
MCKINNON, N. E.: Educating the public about cancer, 544
MACKINNON, William Donald, obituary of, 442
MACKINTOSH, J.: Night pain with fractures, 273
MCLAREN, H. C.: The involution of the cervix, 347 (O); (correspondence), 655
MCLAUGHLIN, C. R.: *Plastic Surgery: An Introduction for Nurses*, 1339
MCLEAN, E. K. *See* WILLIAMS, D. O., 580
MCLEAN, I. E. D.: Whooping-cough immunization, 714
MACLEAN, K. S. *See* KNOX, R., 1081
MACLENNAN, H. R.: Social trends and home confinements, 1353
MACLEOD, E. K. *See* CAUGHEY, J. E., 1216
MACLEOD, Iain N., appointed Minister of Health, 1080
MCLEOD, W. J. M.: Pink disease and infections in infancy, 605
MACMANUS, D.: Improving general practice, 1030
MCMICHAEL, J.: Mechanism of postural hypotension with methonium, 598
— Management of hypertension, 933 (O); 962 (L); 1083 (C), 1190 (C), 1191 (C), 1247 (C), 1248 (C)
MCMURRAY, J. *See* BRODRIBB, H. S., 1060
MCNEE, Sir John: Infective hepatitis: A problem of world health, 1367 (O); 1399 (L)
MCNEIL, C. K.: Combination of congenital anomalies, 762
MCNEISH, W. W. W., and STEWART, C.: Outbreak of Bornholm disease in West Fife practice, 744 (O)
MCSWEENEY, John Leo, obituary of, 389
MAEGRAITH, B. G.: Clinical effects of exposure to heat and sunlight; 1. Heat hyperpyrexia and thermogenic anhidrosis, 1345; 2. Heat exhaustion and sunburn, 1402
MAGEE, H. E. *See* BERRY, W. T., 410
MAGNER, J. W.: Post-mortem and ante-mortem caesarean section, 1092
MAGONET, A. P.: Hypnosis and asthma, 326
— Hypnotism in medicine, 922
Makerere Medical School, history of, 158
MALARIA:
Chemotherapy and chemoprophylaxis; clinical trials in 500 cases and mass prophylaxis in a hyperendemic area (R. N. Chaudhuri, N. K. Chakravarty, and M. N. R. Chaudhuri), 568 (O); 590 (A); (correspondence), 1134
"Daraprim" (B.W. 50–63); new antimalarial; trials in human volunteers (L. G. Goodwin), 732 (O); (correction, 870); 1075 (A)
— study of its effects in falciparum and quartan infections in West Africa (I. A. McGregor and D. A. Smith), 730 (O)
Eradication project in Formosa, 1141
Malaria: Basic Principles Briefly Stated (P. F. Russell), 1395
MALARIA (*continued*):
Mosquito Behaviour in Relation to Malaria Transmission and Control in the Tropics (R. C. Muirhead-Thomson), 203
Proguanil-resistance in Malayan strains of *Plasmodium vivax* (T. Wilson, D. S. Munro, and D. R. Richard), 564 (O); 590 (A)
Subtertian, observations on (W. E. Laufer), 1281 (O)
Treatment, 765
— by intramuscular quinine, 104, 220, 491
Malaya: Diseases among Army personnel, 142
MALLESON, Joan: Sexual disorders in women, 437
— Family planning, 970
Malta: Mass B.C.G. vaccination campaign in 1950 (V. Zammit-Tabona), 837 (O)
Manchester Medical Society, 1039
Section of General Practice; inaugural meeting, 555
Manchester University. *See* Universities: Manchester, 53
Manchurian fever in Korea, 352
MANDEL, L. *See* KERSLEY, G. D., 438, 603
Manipulation: *Treatment by Manipulation*, 2nd ed. fully revised (H. J. Burrows and W. D. Coltart), 1233
MANN, B. T.: Tuberculosis in mental hospitals, 1228 (O)
MANSELL, E. J. B. *See* HART, F. D., 1273
— *See* KEIDAN, S., 1390
MANSON-BAHR, P. E. C.: Megaloblastic anaemia in Indians, 220
Manures, organic, value of, 1357
MARCUS, R.: Progressive bacterial synergic gangrene of the legs, 1230
MARKHAM, Mary. *See* MATHESON, W. J., 742
MARQUAND, H. A.: Paying for the Health Service, 272
MARRACK, J. R. *See* PANTON, P. N., 261
Marriage guidance, 866
— *Marriage: A Book for the Married and about to be Married* (K. Walker), 1178
— *Patterns of Marriage. Study of marriage relationships in the urban working classes* (E. Slater and Moya Woodside), 91
— Royal Commission on Marriage and Divorce; oral evidence on behalf of B.M.A., 1242; 1237 (L)
MARSDEN, J. P., and COUGHLAN, W. J.: Case of confluent chicken-pox with haemorrhagic symptoms, 1066
MARSH, F.: Treatment of tropical ulcers, 220
— Infective gangrene of mouth, 439
— Balantidiasis in South Persia, 1357
MARSHALL, J., and WHITTY, C. W. M.: Value of pneumoencephalography in diagnosis of fits, 847 (O); 1194 (C)
MARSHALL, J. B.: Night pain with fractured clavicle, 163
— What doctors prescribe, 545
MARSHALL, S., and MILLIGEN, K. S.: Unusual features in fatal case of infectious mononucleosis, 1325 (O)
"Marsilid," 858
MARSON, F. G., and MEYNELL, M. J.: Polycythaemia and myelosclerosis, 113 (O)
MARTIN, Charles, obituary of, 1033
MARTLAND, Edward Norman Platt, obituary of, 1137
MARTYN, Frank de Récourt, obituary of, 717
MARTYR, B. *See* GLANVILL, A. T. H., 329
MASEFIELD, W. G.: Admissions to mental hospitals, 921
MASON, A. A.: Hypnotism in medicine, 546
MASON, H. A. C.: Sexual disorders in women, 104
MASON, J. C. *See* GUERRIER, S. M., 1329
MASSIE, E. *See* LIPMAN, B. S., 1287
Mastitis, staphylococcal, in cows as cause of food poisoning in man, 1144
Maternity. *See also* Labour; Obstetrics; Pregnancy
— beds (parliamentary note), 824
MATHESON, W. J., and MARKHAM, Mary: Infantile cortical hyperostosis, 742 (O)
MATTHEWS, B. H. C., elected Professor of Physiology, Cambridge University, 280
MATTHEWS, J. C.: Forgotten tourniquet, 437
MATTHEWS, J. D. *See* ROLLAND, C. F., 1220
MAUDSLEY, R. H.: Case of myositis ossificans progressiva, 954 (O); 1357 (C), 1410 (C)
MAXCY, K. F.: *Rosenau's Preventive Medicine and Hygiene*, 7th ed., 1338
MAXWELL, R. D. H. *See* CAMPBELL, A. J. M., 251
MAXWELL, W. M.: Mr. Bevan, 974
MAY, C. R.: Wooden heel for walking plasters, 1300
MAY, H. B.: Quaternary ammonium antiseptics, 1260
MAYER-GROSS, W. *See* GILLIES, H., 527
MAYRHOFER, O. K.: Self-experiments with succinylcholine chloride, new ultra-short-acting muscle relaxant, 1332 (O); 1342 (A)
MEADE, B. W. *See* ABBOTT, J. D., 523
Measles; graphs (vital statistics), 170, 445, 499, 553, 610
— immunization, 269
Meckel's diverticulum, bleeding; report of two cases (J. N. Walton and Norma D. Lill), 88 (O); (correspondence), 221
Medical and Biological Illustration, 98 (A); 1293 (A)
Medical Art Society's annual show, 929
Medical Artists' Association, 228
Medical Association for the Prevention of War, 612; first national conference, 1089
Medical education. *See* Education, medical
MEDICAL PRACTITIONER:
Attendance at courts, 50
The Doctor. His Career. His Business. His Human Relations (S. R. Truman), 586
Incidence of coronary heart disease in (J. N. Morris, J. A. Heady, and R. G. Barley), 503 (O); 535 (L); (correspondence), 651
Professional prospects; comparison of the position of doctors with that of other professional people (R. Lewis), 615 (O)
Unpunctual doctors (parliamentary note), 1412
Medical Register, appeal against G.M.C. decision to erase name, 338
Medical research. *See* Medical Research Council; Research, medical
MEDICAL RESEARCH COUNCIL:
A.C.T.H. and cortisone in treatment of blood disorders; preliminary report by panel on haematological applications of A.C.T.H. and cortisone, 1261 (O)
Dental Research Unit, 549
Earl of Limerick appointed chairman, 227
Fellowships, 56, 445
Memorandum No. 19: *The Rh Blood Groups and their Clinical Effects*, 667; 870 (C)
Prevention of streptomycin resistance by combined chemotherapy; M.R.C. investigation, 1157 (O); 1179 (L); (correspondence), 1302
Special Report Series, No. 273: Infective hepatitis (F. O. MacCallum and others), 1399
Travelling fellowships in ophthalmology and otology, 172
Tuberculin Survey Committee, 1397 (L)
Unit for research on experimental pathology of the skin, 929
Medical Society of London: 172nd anniversary dinner, 611, 1343
Medical Women's Federation: Memorandum on family planning, with particular reference to contraception, 595, (correspondence), 708, 766, 970, 1027, 1132, 1247, 1354
Medicinal dyes, removal of stains due to, 1366
MEDICINE:
Annual Review of Medicine, vol. 2 (Editor: W. C. Cutting), 205
Fouling the nest, 974, 1195
History: *See also* Biography; Nova et Vetera
— *Advance to Social Medicine* (R. Sand), 1394
— *History of the General Hospital near Nottingham* by F. H. Jacob (V. Z. Cope) (Nova et Vetera), 272
— International History of Medicine Society: 13th congress, 878
— *Story of the Growth of Nursing as an Art, a Vocation, and a Profession* (Agnes E. Pavey), 311
Medicine of the Year, 1951 edition (Editor: J. B. Youmans), 205
Principles of Internal Medicine (Editors: T. R. Harrison and others), 364
Research. *See* Medical Research Council; Research, medical
Textbook of Medicine, 8th ed. (Editors: R. L. Cecil and R. F. Loeb), 748
Tropical. *See* Tropical medicine
MEDICO-LEGAL:
Attendance of doctors at courts, 50
Bequests: American legacy to nationalized hospitals, 1305
— Royal College a "charity," 979
— to nationalized hospitals; complex problems, 1200; 1251 (C)
Blood transfusion, death from toxaemia after, 1036
Breadcrust, fatal, 1361
Conspiracy action against I.C.I. fails, 1139
Damages for personal injuries, 605
Death after 19 years from tooth in lung, 718
Divorce for incurable insanity, 52
Doctor's action against ex-partners fails, 1412
Explosion during anaesthetic, 168; (correspondence), 273, 820, 870, 972, 1083
Hospital beds; medico-legal aspect, 496
Human Blood Groups Utilized in Disputed Paternity Cases and Criminal Proceedings (P. H. Andresen), 1395
Hypnosis; damages against stage hypnotist, 823
Leith hospital funds, 926
Mental condition of witnesses, 1254
"Negligence" in hospital, 1341 (L)
On giving evidence, 644 (A)
Pendulum swinging, 665
Private patient's fees, 551
Refusal to include opticians in list, 335
Responsibility and newly registered practitioners, 1406
Restrictive covenant appeal dismissed, 111
Tourniquet, forgotten, 225; 328 (C), 437 (C), 605 (C)
Tuberculosis contracted by nurse, 443
Xylocaine injection, fatal, 280; 714 (C)
MEDUNA, L. J.: *Carbon Dioxide Therapy. A Neurophysiological Treatment of Nervous Disorders*, 1072
MEDVEI, V. C.: Exophthalmos, 49
Megacolon; death following a barium enema in a child with Hirschsprung's disease (A. Jolleys), 692 (O); (correspondence), 866
Meigs's syndrome: case (C. Nwokolo), 905; 1031 (C)
MELGRAVE, M.: Food and population, 1134
MELLANBY, Sir Edward, Swedish decoration conferred on, 283

MELLANBY, Helen. *See* MELLANBY, Lady May, 7
MELLANBY, Lady May, and MELLANBY, Helen: Dental disease among 14-year-old London school-children in 1947 and 1950, 7 (O)
Memory, impaired, Indian remedies for, 222
Menaphthone, effect in hypoprothrombinaemia, 414
Ménière's disease, value of histamine skin test in, 869
Meningitis associated with congenital dermal sinus, 221
— cerebrospinal, epidemiology of, 211
— tuberculous, deafness with use of dihydrostreptomycin in (J. T. Naismith), 796 (O)
— — treatment with streptomycin and *para*-aminosalicylic acid (S. R. Jamieson), 83 (O); (corrections, 165, 223); 329 (C)
Meningococcal infection; graphs (vital statistics), 170, 282, 336, 392, 981, 1038, 1088, 1141, 1202, 1256, 1414
MENLEY and JAMES: Registered drug samples, 110
MENNIE, A. T.: Cost of treatment, 712
Menopause: Keratoderma climactericum, 1206

MENSTRUATION:
Bacteriology of vaginal flora after use of internal tampons (Elizabeth N. Brand), 24 (O)
Disorders, hormone treatment of, 286
Dysmenorrhoea, ovarian, treated by ovarian denervation (D. C. Wiseman and W. M. Thomas), 141 (O); (correspondence), 272
Effect of cortisone on, 401

Mental condition of witnesses (medico-legal note), 1254
Mental defectives; lecture courses, 722
— — schools for defective children, 286
— — temporary homes for, 250
— disease a symptom of physical defect, 971
— — Tuberculosis in mental hospitals (B. T. Mann), 1228 (O)
Mental Health Research Fund, 699 (L); 975 (C)
Mepacrine in skin disease, lichenoid lesions following, 1406
Mepyramine maleate poisoning, treatment of, 163
— — skin eruption due to, 1405
Mercurial diuretics, contraindications to, 931
Mercury nephrosis in young children, with special reference to teething powders containing mercury (Vera K. Wilson, M. L. Thomson, and A. Holzel), 358 (O); 370 (A); 484 (C), 931 (C)
— poisoning, chronic, in latent finger-print development (G. Forbes and J. White), 899 (O); (correspondence), 1131, 1406
MEREDITH, Lida Orem, obituary of, 871
Metabolism: Changes in basal metabolism, serum-protein-bound iodine, and cholesterol during treatment of hypothyroidism with oral thyroid and *L*-thyroxine sodium (J. D. Robertson and H. F. W. Kirkpatrick), 624 (O)
— work at University College Hospital, 648, 667
METCALFE, R. H.: Fractures in the aged, 1240 (correction, 1366)
"Methedrine," effect on blood pressure, 106

METHONIUM COMPOUNDS:
Decamethonium iodide; undesirable side-effects (S. M. Guerrier and J. C. Mason), 1329 (O)
Hexamethonium bromide and decamethonium iodide, use with electroplexy (J. D. Montagu), 1336
M. & B. 1863, a homologue of hexamethonium, in treatment of hypertension (S. Locket and others), 254 (O)
Medical sympathectomy with, 597
Oral treatment of hypertension (A. J. M. Campbell, J. G. Graham, and R. D. H. Maxwell), 251 (O)

Methyl androstenediol, 728
Methyl bromide poisoning, 1122 (A)
Metric system in medicine, 819, 924, 1038, 1084
Mexico: Smallpox incidence (vital statistics), 1088
Meyer and Bleuler, 326
MEYER, P.: Coronary disease in doctors, 651
MEYNELL, M. J. *See* MARSON, F. G., 1113
MICHAEL, A. M.: Hypnosis in childbirth, 734 (O); (correspondence), 1132
Microbiology: *Bacterial and Virus Diseases. Antisera, Toxoids, Vaccines, and Tuberculins in Prophylaxis and Treatment*, 2nd ed. (H. J. Parish), 91
— First congress of Hungarian Microbiological Society, 531
— Society for General Microbiology, 1090, 1204
Microfilariae: investigations into cause of periodicity in man (J. A. McFadzean), 1106 (O)
Micro-organisms, catalogues of, 338
Microscopy: *Intelligent Use of the Microscope* (C. W. Olliver), 534
— *Phase Microscopy; Principles and Applications* (A. H. Bennett and others), 1117
Micturation, frequency of; causes and treatment (H. P. Winsbury-White), 372, 547 (C), 549
MIDDLEMISS, J. E.: Hypnotism in medicine, 487
MIDGLEY, R. L. *See* EDWARDS, P. W., 49
Midwives: Central Midwives Board, 983
Migraine, aetiology, 107
— treatment with oestrogen or androgens, 1408
Mikulicz-Radecki, Johann, nationality of, 975
Milk for school children (parliamentary notes), 390
— reabsorption after arrested lactation, 985
— secretion, physiology of, 1340 (L)
MILLARD, Charles Killick, obituary of, 660, 820
MILLER, A.: Salivary tumour of the hard palate, 600
MILLER, J. H.: Pseudocyesis, 816
MILLER, S. J. H.: Symptomatology of congestive and simple glaucoma, 456 (O); 712 (C), 764 (C), 869 (C), 1083 (C), 1356 (C)
MILLER, W. S.: Persistent urinary *Salmonella dublin* carrier with bilharziasis, 628 (O)
MILLER, William Henry, obituary of, 167, 279
MILLIGAN, E. H. M.: Experiment in peacemaking, 818
MILLINGEN, K. S. *See* MARSHALL, S., 1325
MILLS, George Percival, obituary of, 662, 821
MILLS, Robert James, obituary of, 977
MILLS, W. G.: Torsion of fallopian tube, 1407
Mind, Perception, and Science (W. R. Brain), 856
Mining: Colliery medical services (parliamentary note), 978
Minister of Health, Iain N. Macleod appointed, 1080

MINISTRY OF HEALTH:
Annual report, 335
Appointments, 46
21st annual exhibition of the Art Club and Camera Club, 228
Change of address, 611

Minnitt's gas-air apparatus, portability, 328
"Miracil D." *See* "Nilodin"
MITCHELL, G. P.: Early treatment of poliomyelitis, 649; 923 (C)
MITCHELL, J. S., elected F.R.S., 703
MITCHELL, J. V.: Introducer for plastic cannulae (preparations and appliances), 435
Mitral stenosis: Valvotomy; further reports on 100 cases (C. Baker and others), 1043 (O); 1073 (L)
— valve disease in the light of surgical treatment (G. Bourne), 896 (O)
MODEL, A.: What doctors prescribe, 600
MOIR, L. J.: Treatment of migraine with oestrogen or androgens, 1408
MOLLISON, P. L.: *Blood Transfusion in Clinical Medicine*, 472
Molluscum contagiosum, treatment, 57
MONCRIEFF, A., and WALTON, A. M.: Visiting children in hospital, 43
Mongolism associated with congenital duodenal obstruction (M. Bodian and others), 77 (O); (correspondence), 276, 384
Mongols, congenital defects in, 276
Moniliasis due to antibiotic therapy, 523, 761
Mononucleosis, infectious. *See* Glandular fever
MONRO, A. B. *See* HURLEY, M. J., 1027
MONRO, T. K.: *The Physician as Man of Letters, Science, and Action*, 959
MONTAGU, J. D.: Use of hexamethonium bromide and decamethonium iodide with electroplexy, 1336
MONTESSORI, Maria, obituary of, 1085; tribute to, 1304
MONTUSCHI, E.: Treatment of moniliasis, 761
MOODIE, W.: Specific language disability, 325
MOOR, F.: What doctors prescribe, 436
MOORE, C. V. (Editor-in-Chief): *Proceedings of the Third International Congress of the International Society of Hematology*, 1072
MOORE, R. *See* KELLGREN, J. H., 181, 997
MOORE, T.: Prostatic obstruction and inguinal hernia; a new approach, 362; 547 (C)
MOOTATAMBY, N., obituary of, 51
MORAN, Lord, address at Royal Sanitary Institute Health Congress, 1021; 1196 (C), 1246 (C)
Morbidity: Sickness studies from insurance statistics, 753 (A)
MORGAN, John Patrick, obituary of, 1359
MORGAN, W. T. J., appointed Professor of Biochemistry, London University, 336
MORGANS, M. E. *See* BARNS, H. H. F., 1058
MORISON, J. E.: *Foetal and Neonatal Pathology*, 639
MORLAND, A.: Isonicotinic hydrazide as wonder drug, 970
Morphine, idiosyncrasy to, 502
MORRICE, J. K. W.: Are neurotic symptoms imagined?, 326
MORRIS, J. N., HEADY, J. A., and BARLEY, R. G.: Coronary heart disease in medical practitioners, 503 (O); 535 (L); (correspondence), 651
— *See* LOGAN, W. P. D., 1190
MORRIS, J. V.: Indian remedies for poor memory, 222
MORRISON, R. J. G., and BAIRD, J. P.: Orchitis in Bornholm disease, 198 (O)
MORTON, A. G.: *Soviet Genetics*, 472
Mosquitoes: *Mosquito Behaviour in Relation to Malaria Transmission and Control in the Tropics* (R. C. Muirhead-Thomson), 203
MOSSBERG, H.: Vitamin K in the treatment of acute hepatitis, 1382 (O)
Mothers, unmarried, homes for, 828
Motor-cars by hire-purchase for doctors (parliamentary note), 1087
— for doctors, 609
— petrol tax, 644 (A)
Motor-cycle accidents, 1183 (A)
MOTTRAM, R. F. *See* COOPER, K. E., 1409
MOULDEN, G. A.: Tooth in tonsil, 766
Mountaineering, medical problems of, 1120 (L)
Mouth: Aphthous stomatitis treated with local aureomycin, 1082, 1246
— gangrene in African children, 198; 439 (C), 975 (C)
Mucus extractor for use on newborn infant (H. H. F. Barns), 598
MUGGLETON, P. W. *See* UNGAR, J., 1211
MUIRHEAD, William Martin, obituary of, 717
MUIRHEAD-THOMSON, R. C.: *Mosquito Behaviour in Relation to Malaria Transmission and Control in the Tropics*, 203
Mulago Hospital, Uganda, history of, 158
Mulvany's operation for stress incontinence, 1384
Mumps, immunity in, 912 (A)
— immunization, 270
— with involvement of lacrimal glands; case (J. F. Galpine and J. Walkowski), 1069; 1250 (C)
MUNRO, D. S. *See* WILSON, T., 564
MURDOCH, D.: Tuberculosis and surgical abortion, 1030
MURPHY, E. F. *See* SWEETNAM, W. P., 465
MURRAY, Herbert Edward, obituary of, 279
MURRAY, N.: Hypnosis in childbirth, 1132
MURRAY, William Alfred, obituary of, 925
MURRELL, Marjorie. *See* BATCHELOR, R. C. L., 365
Muscle relaxants. *See also* Gallamine triethiodide; Methonium compounds; Succinylcholine, Tubocurarine
— — effect on blood pressure, 492
— — Ultra-short-acting (H. Richards and H. R. Youngman), 1334 (O); 1342 (A)
Muscles: Radiological changes in skull in dystrophia myotonica (J. E. Caughey), 137 (O)
— testing for serratus anterior weakness (Cynthia M. Walker), 1393
MUSHIN, W. W.: Anaesthesia for minor procedures, 431; 711 (C)
Mycology: Organization for medical mycology, 1400 (A)
Myeloid metaplasia, splenogram in, 988
Myelomatosis, multiple, aetiology, treatment, and prognosis of, 340, 558
Myelosclerosis, idiopathic, splenogram in, 988
— with polycythaemia (F. G. Marson and M. J. Meynell), 1113 (O)
Myocarditis, toxic, effect of digitalis on, 1205
Myopia, 320
Myositis, epidemic. *See* Bornholm disease
— ossificans progressiva; case (R. H. Maudsley), 954 (O); 1357 (C), 1410 (C)
— — treatment with parathyroid suggested, 1357, 1410
MYRBÄCK, K. *See* SUMNER, J. B., 423

N

Naevi in pregnancy, 162
NAGLEY, M. M.: Isonicotinic acid hydrazide, 1133
Nails, white spots on, 985
NAISH, F. Charlotte: Treatment of acidosis, 1196
NAISH, J.: Treatment of anaemia, 599
NAISMITH, J. T.: Deafness with use of dihydrostreptomycin in tuberculous meningitis, 796 (O)
Napalm bomb, 818, 1031
α-Naphthylamine; conspiracy action against I.C.I., 1139
Naphthylamines as cause of bladder tumours, 1123 (A)
Nasal sinuses, lymph draining of, 118
NASH, D. F. E. *See* HIGGINS, T. T., 695
NATHAN, Nestor John Stanley, obituary of, 110
National Council of Social Service: *Voluntary Service and the State, Study of the Needs of the Hospital Service*, 367 (L); (correspondence), 657
National Formulary, 1952, 701 (A); 924 (C)
— — and proprietary drugs (parliamentary note), 1361

NATIONAL HEALTH SERVICE:
Appliances supplied in 1951 (parliamentary note), 552
Bevan versus Spens, 1291 (A)
Bevan's book, 749 (L); (correspondence), 870, 974, 1030
Capitation fee; average for year ending 31 March (parliamentary note), 875
Central pool, 824, 1139
Charges for dental treatment (parliamentary note), 978
— for prescriptions and appliances, 283, 665, 719, 772, 1181 (A)
— — — collection by slot-machines (parliamentary note), 1413
— for services, 312 (L); 333 (parliamentary note); 749 (L); 772 (parliamentary note)
Clinical research in the National Health Service (Sir Cecil Wakeley), 917; 974 (C), 1030 (C), 1196 (C)
Co-operation; report of Central Health Services Council, 912 (A)
Debate in the Lords (parliamentary note), 770
Doctor's lists, numbers of patients on (parliamentary note), 1139
Economics and doctors: the cost of the Health Service, 642 (L); (correspondence), 818
Effect of the "easy approach" on morale, 1343 (A)
Efficiency of the Health Service, 335
Estimated savings, 1361
Estimates, 497 (parliamentary note); 701 (L)
Expenditure, 759
The Health Services. A compendium of conference papers with additional chapters published for the Institute of Public Administration, 803
Hospitals: Compulsory retirement of staff, 38 (A)
Isle of Man, 927
National Formulary, 1952, 701 (A); 924 (C); 1361

NATIONAL HEALTH SERVICE (*continued*):
National Health Service Bill; second reading, 771, 978; committee stage, 873; report stage, 1034; in the Lords, 1086, 1138, 1199 (parliamentary notes)
Paying for the Health Service, 272, 437
Payments to general practitioners (parliamentary note), 824
Prescriptions; average cost, 1940–51 (parliamentary note), 824
— charges. *See under* Charges, *above*
Rayon surgical dressings, 1361
Reciprocal arrangements with foreign, Dominion and Colonial Governments (parliamentary note), 1200
Refusal to include opticians in list (medico-legal note), 335
Remuneration, adjudication, 749
— — preparing the case, 263 (L)
— — terms, 697 (L); 718, 771, 824 (parliamentary notes)
— Award and the Working Party, 1235 (L)
— Central pool; composition of Working Party (parliamentary note), 1139; terms of reference and recommendations, 1200
— — — redistribution negotiations (parliamentary note), 824
Role of statistics in regional hospital planning, with special reference to Birmingham Hospital Region (Vera Norris), 129 (O); (correspondence), 272, 437
National Insurance: Advisory Committee (parliamentary note), 1200
— — allowances and pensions (parliamentary note), 1087
— — benefits for long-term hospital in-patients, 1080
Nature cure, 653, 764, 818, 1031
NAYAR, P. T. K.: Tuberculous glands in the neck, 830
"Negligence" in hospital, 1341 (L)
NEILAN, Charles James, obituary of, 1198
NEILL, Andrew Gordon Currie, obituary of, 551
NELSON, G.: Schistosomiasis in the West Nile District, 158
NELSON, M. G.: Purpura and pancytopenia complicating arsenotherapy, 300 (O); 714 (C)
Nephrosis in young children due to mercury, with special reference to teething powders containing mercury (Vera K. Wilson, M. L. Thomson, and A. Holzel), 358 (O); 370 (A); (correspondence), 484, 931
NERVES: *See also* Neuralgia; Neuritis; Neurology; Neurophysiology; Sympathectomy
Anterior interosseous nerve, isolated neuritis of (L. G. Kiloh and S. Nevin), 850 (O)
Fifth and seventh cranial nerves in relation to nervous mechanism of taste sensation: a new approach (W. Harris), 831 (O); 860 (A)
Ganglion block, autonomic, during anaesthesia, 1144
— — indications and technique, 777
Sympathetic nerve endings, enzyme at (J. H. Burn), 784 (O); 861 (A)
Netherlands: Decorations awarded, 1257
NEUBUERGER, K. T.: *Atlas of Histologic Diagnosis in Surgical Pathology*, 748
Neuralgia, post-herpetic, treatment of, 230
Neuritis, isolated, of anterior interosseous nerve (L. G. Kiloh and S. Nevin), 850 (O)
— peripheral, in systemic lupus erythematosus (R. H. Heptinstall and G. S. C. Sowry), 525 (O)
Neurology: *Anatomy of the Nervous System*, 2nd ed. (O. Larsell), 1010
— *Medical Neuropathology* (I. M. Scheinker), 639
— *Synopsis of Neurology* (W. F. T. Tatlow, J. A. Ardis, and J. A. R. Bickford), 1010
Neurophysiology: *Mind, Perception, and Science* (W. R. Brain), 856
Neurosis: Are neurotic symptoms imagined?, 326
Neurosurgery: *Frontal Lobotomy and Affective Behavior; A Neurophysiological Analysis* (J. Fulton), 1394
— *see also* Sympathectomy
NEVILLE, N. A. *See* YELL, W. L., 1302
NEVIN, S. *See* KILOH, L. G., 850
New Guinea: Nutrition, 37 (A)
New Year Honours, 44 (correction, 118)
Newborn. *See* Infant, newborn
NEWMAN, N. H. *See* HOBSON, A. C. S., 1111
NICHOLSON, J. C. *See* DEVITT, D. C., 1303
NICKLIN, Reginald, obituary of, 279
NICKOLLS, L. C.: Death on the roads, 47
NICOLL, J. A. V.: Spontaneous rupture of the spleen, 801
Nicolle, Charles, foundation in commemoration of, 144
Nicotinic acid hydrazides in treatment of tuberculosis, 858 (L); 868 (C), 970 (C); 983; 1015 (A); 1028 (C), 1081 (C), 1133 (C); Ministry of Health statement, 775
1-*iso*Nicotinyl-2-isopropyl hydrazine in treatment of tuberculosis, 478
Nidoko disease, 477
Nigeria: Sickle-cell trait in Western Nigeria: Survey of 1,881 cases in the Yoruba (D. B. Jelliffe and J. Humphreys), 405 (O); 426 (L); (correspondence), 763, 920
— Southern, blindness in (G. E. Dodds), 584; 1248 (C)
Night nurses' paralysis, 557
— sweats; investigation and treatment, 932
"Nilodin" in treatment of *Schistosoma haematobium* (M. A. Haseeb), 1331 (O)
Nitrogen mustard analogues, 1343
NIXON, W. C. W.: Family planning, 708
Nobel Prize Winners' Convention, 1258
Nomenclature: Debauchery of honest words, 548, 762, 924, 1084
— Dermatologist's protest, 1406
— Latin, is it dead? 924, 1084, 1197, 1304
— Medical semantics, 813, 870
— of diseases and operations, 330, 492, 715
L-*Nor*adrenaline. *See* Adrenaline
NORRIS, Vera: Role of statistics in regional hospital planning, with special reference to Birmingham Hospital Region, 129 (O); (correspondence), 272, 437
North of England Obstetrical and Gynaecological Society, 722, 828, 1203; annual general meeting, 393
Nose bleeding, 1124 (A); 1197 (C)
— granulomatous ulcer treated with chloramphenicol (I. B. Sneddon and J. Colquhoun), 298 (O); 491 (C)
— instrumentation, analgesics for, 285
NOTES AND COMMENTS: 118, 174, 230, 340, 396, 448, 502, 558, 614, 670, 724, 778, 830, 880, 932, 1042, 1092, 1144, 1206, 1312, 1366, 1418
Notification of disease, 474 (L); 713 (C)
Nottingham: *History of the General Hospital near Nottingham* by F. H. Jacob (V. Z. Cope) (Nova et Vetera), 272
NOVA ET VETERA:
Apologie and Treatise of Ambroise Paré, edited by G. Keynes (C. Singer), 1129
Caelius Aurelianus; On Acute Diseases and on Chronic Diseases, edited by I. E. Drabkin (C. Singer), 433
English Pioneers of Anaesthesia (Beddoes, Davy, and Hickman) by F. F. Cartwright (C. Singer), 706
The Essays of Jean Rey. A facsimile reprint of the original edition of 1610, *with an introduction by Douglas McKie* (C. Singer), 1188
History of the General Hospital near Nottingham by F. H. Jacob (V. Z. Cope), 272
Seventeenth Century Doctor and his Patients; John Symcotts, 1592?–1662 (F. N. L. Poynter and W. J. Bishop), Bedfordshire Historical Record Society Publications, Vol. XXXI (C. Singer), 1350
Translation of Galen's Hygiene (De Sanitate Tuenda) by R. M. Green (C. Singer), 1404
William Davidson of Aberdeen. The first British Professor of Chemistry by J. Read (C. Singer), 380
"Novalgin" as cause of agranulocytosis, 1273, 1292
NURSES AND NURSING:
Guide to Professional Nursing, 960
National Hospital Service Reserve, 17
Night nurses' paralysis, 557
Nursing fellowship at Edinburgh, 1257
Story of the Growth of Nursing as an Art, a Vocation, and a Profession, 3rd ed. (Agnes E. Pavey), 311
Tuberculosis contracted by nurse; award of industrial injury benefit (medico-legal note), 443
NUTRITION: *See also* Food; Vitamins
Effect of liver supplement on growth of children (J. Yudkin), 1388 (O); 1401 (A)
French summer schools, 1258
Geography of Hunger (J. de Castro), 1234
In New Guinea, 37 (A)
International Union of Nutritional Sciences symposium, 1364
Intravenous feeding with fat emulsions, 1400 (A)
Need for better food (parliamentary note), 334
Nutrition Society: Conference on vitamin B_{12}, 160; officers elected, 1204
Nutrition Work in Greece (A. G. Tsongas), 145; 384 (C)
Nutritional Improvement of Life (H. C. Sherman), 145; 384 (C)
Status of Arab refugees from Palestine, 1285
Teaching Better Nutrition (Jean R. S. Ritchie), 145; 384 (C)
NWOKOLO, C.: Case of Meigs's syndrome, 905; 1031 (C)
Nyasaland: Construction of new hospital begun, 1364
"Nydrazid," 858

O

Oak, sensitivity to, 1092
OAKLEY, C. L., appointed to Brotherton chair of bacteriology, University of Leeds, 1255
OAKLEY, D. E.: Is penicillin dangerous? 713
OAKLEY, W., and PEEL, J.: Diabetes and pregnancy, 1248
OBITUARIES:
Ainsworth, Sir Ralph Bignell, 331
Allen, Robert Hugh, 1034
Amsler, Albert Maurice, 1412
Baird, William Joseph McLearn, 224
Bishop, Thomas Henry, 664
Bloch, Carl Edvard, 663
Boyde, Harry Caine, 926
Braithwaite, Eldred Curwen, 389, 718
Bramwell, Edwin, 768, 821
Brauer, Ludolph, 51
Brown, Samuel Stuart, 496
Brown, William, 1136
OBITUARIES (*continued*):
Brownlie, Andrew, 111
Bullough, Fred, 823
Carden, William Alfred, 111
Clifford, John Hudson, 1252
Coullie, Alexander Glover, 925
Crellin, Douglas, 1252
Cutler, Frederick John, 1086
Dakin, Henry Drysdale, 441
Dobriner, Konrad, 715
Dodgson, Robert William, 871
Dundon, John, 663
Dunlop, James (Glasgow), 822
Dutton, Edward Charles, 1359
Elford, William Joseph, 495
Ellison, Philip Oswald, 871
Feddon, Walter Fedde, 662
Ferguson, Joshua, 167
Findlay, George William Marshall, 658, 715, 769, 822
Fleming, Geoffrey Balmanno, 977
Fletcher, Nigel Corbet, 223
Forbes, William John, 1305
Fraser, Robert Moore, 496
Freudenthal, Walter, 820
Gaskell, Sir Arthur, 278
Gaston, Joseph, 822
Gleave, Humphrey Hart, 925
Gordon, Thomas Landles, 52
Gowans, Francis Jollie, 926
Graham, Frederick Montague, 550
Grattan, Henry William, 1411
Gregory, Hazel Howard Chodak, 278
Greig, John Isdale, 332
Gunesekera, Sir Frank Arnold, 494
Hall, Charles Herbert, 717
Hare, John, 1198
Hartmann, Henri, 167
Hartmann, Max, 389
Harwood, Leonard Austin, 1252
Haughton, William Steele, 331
Heseltine, Michael, 663, 716, 822
Hill, Sir Leonard Erskine, 767
Howard-Jones, John, 978
Hugo, Edward Victor, 224
Ibbotson, William, 1359, 1411
Jackson, Henry Law, 1360
Kennedy, Robert Foster, 165, 223
Kilroe, Laurence, 1197, 1252
Kingston, William George, 1198
Lace, Frederick, 1198
Ledgard, Herbert Arthur, 1033
Leslie, Leonard, 872
Levin, Joseph John, 1085
Logie, John William, 550
Lowe, Godfrey John Ralph, 1032
Luce, Sir Richard Harman, 493, 662
Macbeth, Archibald, 332
MacEwan, Archibald, 550
McGibbon, John Paton, 1031
Mackie, Alexander Hendry, 1033
Mackinnon, William Donald, 442
McSweeney, John Leo, 389
Martin, Charles, 1033
Martland, Edward Norman Platt, 1137
Martyn, Frank de Récourt, 717
Meredith, Lida Orem, 871
Millard, Charles Killick, 660, 820
Miller, William Henry, 167, 279
Mills, George Percival, 662, 821
Mills, Robert James, 977
Montessori, Maria, 1085
Mootatamby, N., 51
Morgan, John Patrick, 1359
Muirhead, William Martin, 717
Murray, Herbert Edward, 279
Murray, William Alfred, 925
Nathan, Nestor John Stanley, 110
Neilan, Charles James, 1198
Neill, Andrew Gordon Currie, 551
Nicklin, Reginald, 279
O'Neill, Charles Sefton, 442
Overend, Thomas Dallas, 872
Parkinson, Alfred Howard, 550
Parsons, Alfred Robert, 549, 664
Peace, Percy Clift, 224
Pearson, Henry Carden, 717
Penman, John, 1305
Pentony, John Joseph Kevin, 1198
Phemister, Dallas Burton, 166
Phillips, John, 225
Purvis, John Allen Spottiswoode, 332
Pollard, Walter Henry, 389
Ravenhill, Thomas Holmes, 1032
Rees, Sir John Milsom, 975, 1137
Reid, Henry Charles Cecil, 871
Renshaw, Graham, 389
Rinkel, Lambert Ronald Joslin, 51
Robbins, Frank Hubert, 495
Russell, Albert Victor John Charles, 389
Shaw, Raymond Henry, 551
Sherrington, Sir Charles Scott, 606; 589 (L)
Shields, Sir Douglas Andrew, 549
Skeels, Percy Bernard, 225
Skevington, Sir Joseph Oliver, 660
Smith, Edward Davison, 717
Smith, Frederick Hugh, 279
Smith, James Adam, 279
Square, William Holberton, 111
Stallard, John Prince, 332
Steven, Peter Alexander, 1199
Stock, William Stuart Vernon, 1411

OBITUARIES (*continued*):
Symes-Thompson, Henry Edmund, 278
Talbot, Ernest, 872
Taylor, Frederick William, 823
Taylor, John Frank, 1410
Thornton, George Lestock, 110, 332
Vedder, Edward Bright, 441
Wales, Edward Garneys, 1305
Wallace, Sir David, 976
Wallis, Preston Richard, 442
Wharton, John, 1136
White, Gilbert Benjamin Mower, 1086
Wilson, Alexander Garrick, 51
Young, David Hunter Dow, 871

O'BRIEN, P. J.: Placing lymph for vaccination, 385

OBSTETRICS: *See also* Abortion; Caesarean section: Labour; Pregnancy; Puerperium; Royal College of Obstetricians and Gynaecology
Exercises Before Childbirth (Kathleen Vaughan), 1118
Priority for primigravidae in hospitals, 1022
Textbook of Obstetrics (J. F. Cunningham), 534

Occupational medicine. *See* Industrial medicine
O'CONNOR, J. A.: Hypnotism in medicine, 487
O'CONNOR, W. A.: Physical therapy versus psychotherapy, 1250
O'DONOVAN, D. K. *See* O'MEARA, R. A. Q., 605
Oedema following cortisone therapy, 401
Oesophagectomy; plastic artificial gullets, 1358
Oesophagitis (J. B. Harman), 941 (O); 961 (L); 1250 (C)
— personal experience of, 1250
Oesophagus, carcinoma: diagnosis and surgical treatment of 159 cases (R. H. Franklin and J. J. Shipman), 947 (O); 961 (L)
— *The Esophagus and Pharynx in Action. A Study of Structure in Relation to Function* (W. Lerche), 639
— fatal perforation of, by bread crust (medico-legal note), 1361
Oestrogen level in pregnancy, 619
Oils, penetrating, dangers from contact with, 829

OLD AGE:
Adding Life to Years (Lord Amulree), 364
Employment of older people, 667, 1409
Fractures in the aged (R. H. Metcalfe), 1240 (correction, 1366)
Hospital admission for the senile (parliamentary note), 874
International Association of Gerontology and rapid progress of gerontology (V. Korenchevsky), 375
Problem of the aged and sick as seen by the general practitioner (M. O'Sullivan), 1297
Scope and problems of geriatrics, 650
Social and Biological Challenge of our Ageing Population. Proceedings of the Eastern States Health Education Conference, March 31–April 1, 1949, 638
Trends in Gerontology (N. W. Shock), 638
Welfare arrangements in Scotland, 542

OLDHAM, J. B.: Penicillin cream dangerous? 545
OLIVER, L. C., and GIBBS, J. R.: Homolateral pyramidal signs with tumours of posterior fossa, 634 (O)
Oliver Memorial Fund fifth award, 1364
OLLIVER, C. W.: *The Intelligent Use of the Microscope*, 534
O'MEARA, R. A. Q., HICKEY, M. D., JESSOP, W. J. E., and O'DONOVAN, D. K.: Scientific exhibition at the Annual Meeting in Dublin, 605
Omentum, angiomatous lesions of, in angioma venosum racemosum (I. Gilbert), 468 (O)
Omnibus drivers, selection of, 150 (A)
Onchocerciasis in West Africa, 853
— treated with hetrazan; histological study (F. Hawking), 992 (O)
O'NEILL, Charles Sefton, obituary of, 442
Oophorectomy, bilateral, psychological effects of, 557
Ophthalmia neonatorum; graphs (vital statistics), 170, 981, 1038, 1088, 1141, 1203, 1256
— phlyctenular, treatment with streptomycin, 122
Ophthalmic services, expenditure on, 760
Ophthalmological Society of United Kingdom, 72nd annual congress, 1112

OPHTHALMOLOGY: *See also* Blindness; Colour vision; Eye; Vision
Course in industrial ophthalmology, 1364
Faculty of Ophthalmologists: officers elected, 1413
Genetics in Ophthalmology (A. Sorsby), 203
Ophthalmology (A. E. Town), 1117
Streptomycin in ophthalmology (A. Sorsby, J. Ungar, and N. L. Bailey), 119 (O); 151 (A)
Systemic Ophthalmology (Editor: A. Sorsby), 310
Travelling fellowships, 172

Opticians: Refusal to include opticians in list (medico-legal note), 335
— *Report of the Interdepartmental Committee on the Statutory Registrations of Opticians*, 1074 (L)
ORAM, S. *See* CLAYTON, S., 1010
Orgasm, absence of sensation during, 1092
ORLEY, A.: Shortage of *x*-ray films, 219
Orthopaedics: *Orthopaedic Nursing* (Mary Powell), 696
— *Regional Orthopedic Surgery* (P. C. Colonna), 146
Orthoptists Section, National Register of Medical Auxiliary Services, 1309
Osler, Sir William, fatal illness of, 156
— *Osler: The Man and the Legend* (W. R. Bett), 1178
Osler Club symposium on recent history of pneumonia, 156
OSMOND, T. E.: Treatment of gonorrhoea, 863
Ossification, accelerated, in cretin caused by high thyroid dosage (T. Colver and T. Lodge), 89
OSTENSO, Martha. *See* KENNY, Elizabeth, 802
Osteoarthritis, generalized, and Heberden's nodes (J. H. Kellgren and R. Moore), 181 (O); 208 (L); 383 (C), 765 (C)
Osteochondritis: Perthes's disease, prognosis in, 1311
OSTLERE, G.: Decomposition of trichlorethylene, 1405
O'SULLIVAN, M.: Problem of the aged and sick as seen by the general practitioner, 1297
Otitis media, flying and bathing in, 777

OTO-RHINO-LARYNGOLOGY:
Atlas der Hals-, Nasen-, Ohren-Krankheiten, 3rd enlarged ed. (C. von Eicken and A. S. van Treeck), 1072
British Association of Otolaryngologists, annual general meeting, 227
Ear, Nose, and Throat Diseases for the General Practitioner (W. McKenzie), 473
Travelling fellowships in otology, 172

OTTLEY, Constance M.: Carcinoma of the stomach, 492
OUTHWAITE, J. E. *See* PIM, D. C.
Ovary: Denervation for ovarian dysmenorrhoea; review of 70 cases (D. C. Wiseman and W. M. Thomas), 141 (O); (correspondence), 272
— Meigs's syndrome; case (C. Nwokolo), 905; 1031 (C)
— Psychological effects of bilateral oophorectomy, 557
OVEREND, Thomas Dallas, obituary of, 872
Overseas Science Students' Association, 929
Ovum, unfertilized, length of survival, 285
OWEN, T. K. *See* WRIGHT, G. A., 800
Oxford University. *See* Universities: Oxford
Oxygen, pure, as cause of anaesthetic explosions, 273

P

PADLEY, R. (Editor): *City of Birmingham Annual Abstract of Statistics. No.* 1. 1931–1949, 908
Paediatrics. *See* Children; Infants
Pain, congenital indifference to (E. K. Westlake), 144
— relief with intravenous procaine, 266 (A)
Palate: *Cleft Lip and Palate* (W. G. Holdsworth), 695
— tumour of, 329, 600
Palsy, cerebral. *See* Paralysis, cerebral
"Paludrine." *See* Proguanil
Pancreas: Islet-cell tumour, 307 (O)
— *Roentgen Manifestations of Pancreatic Disease* (M. H. Poppel), 960
Pancreatitis, acute; unusual case, 1070
Pancytopenia, hypersplenic, splenogram in, 989
PANKHURST, J. H. F.: What doctors prescribe, 485
PANTON, P. N., and MARRACK, J. R.: *Panton and Marrack's Clinical Pathology*, 6th ed., 261
Pantothenic acid, 643 (A)
PAPACOSTAS, G.: Discovery of formol gel reaction, 163
Para-aminosalicylic acid. *See p*-Aminosalicylic acid
Paralysis agitans. *See* Parkinsonism
— cerebral; incidence and prognosis; interim report (Irene M. Holoran), 214
— in night nurses, 557
"Parathion," 543
— poisoning, 1024
Paratyphoid fever vaccination, 270
— *see also* Enteric fever
Paré, Ambroise, Apologie and Treatise of, edited by G. Keynes (C. Singer) (Nova et Vetera), 1129
PARISH, H. J.: *Bacterial and Virus Diseases. Antisera, Toxoids, Vaccines, and Tuberculins in Prophylaxis and Treatment*, 2nd ed., 91
— *See* LAURENT, L. J. M., 1294
PARKINSON, Alfred Howard, obituary of, 550
Parkinsonism; aetiology, diagnosis, prognosis and treatment (H. G. Garland), 153, 382, 490
— diagnosis: Head-dropping test (R. Wartenberg), 687 (O)
— following severe herpes ophthalmicus (G. Strong), 533 (correspondence), 820

PARLIAMENT:
Addison, Lord; tribute in House of Lords, 334
Aden, hospital facilities at, 665
Adjudication award, 718, 771, 824
Agene: Medical Research Council investigations, 824
Agenized bread, 334
Agriculture (Poisonous Substances) Bill, 1035, 1199, 1252, 1360
Alcohol and road accidents, 1413
Arsenic in cigarettes, 824
Artificial limbs, charge for, 552
Bedding, hygienic, 824
Broadmoor; costs of maintenance of patients, 1139
— inquiry, 1086
Burns: prevention, 665
Cancer cure; inquiry into Rees Evans' claims, 824, 1253
— education of public, 824
— Funds for research, 773
Cars by hire-purchase for doctors, 1087

PARLIAMENT (*continued*):
Children: Beds for mentally deficient children, 1413
— cruelty to, 1087
— healthier children, 334
— visiting children in hospital, 824
Cod liver oil for tuberculous patients, 1254
Colliery medical services, 978
Connaught Military Hospital; conditions and accommodation, 497
Corneal grafting: Bill to make provision for use of eyes of diseased persons, 1138, 1200, 1252, 1360
Cremation Bill, 1253, 1360
— certificates, fees for, 334
Cross-infection among children, 1412
Defamation (Amendment) Bill, 266 (A)
Dentists' Bill in the Lords, 390, 609
Dermatitis, industrial, 665
Doctors' pay, 1253
Drugs: Dangerous Drugs Act; reconstitution of tribunal, 824
— prices in advertisements to medical practitioners, 1361
Epileptics, schools for, 391
Food: Statutory requirements for sanitary conditions of sale, 1200
Foot-and-mouth disease, 1087
General practitioner in hospitals, 770, 1087
Haemophilia; Dr. W. A. Timperley's method of treatment, 1413
Hospitals; costs increased, 773
— food costs per patient, 497, 1360
— Staffing costs, 1200
Hypnosis on the stage, 773
Hypnotism Bill, 979
Identity cards abolished; numbers retained for National Health Service, 497
— numbers for temporary residents, 1412
Industrial injuries, 391
Insecticides: Poisonous chemicals in agriculture; House of Lords debate, 1035
International exchange of medical facilities, 1035
Isle of Man: Rising costs of Health Service, 927
Kroll's treatment of brain injuries, 609
Lead poisoning, 1087
London University's Senate challenged, 1199
Maternity beds, 824
Milk for school children, 390
National Formulary and proprietary drugs, 1361
National Health Service: Appliances supplied in 1951, 552
Capitation fee; average for year ending 31 March, 875
Central pool: Composition of working party, 1139
Charges for dental treatment, 978, 1034
— for prescriptions; collection by slot-machines, 1413
— Charges for services, 333, 772
Debate in the Lords, 770
Doctor's lists, numbers of patients on, 1139
Efficiency of the Health Service, 335
Estimates, 497
Payments to general practitioners, 824
Prescriptions; average cost, 1940–51, 824
charges, 665, 772
exemption, 391
Reciprocal arrangements with foreign, Dominion and Colonial Governments, 1200
Remuneration; White Paper on terms of reference and recommendations of Working Party suggested, 1200
National Health Service Bill; second reading, 771
— committee stage, 873
— in the Lords, 1034, 1086, 1138
— report stage, 1199
National Insurance: Allowances and pensions, 1087
National Insurance Advisory Committee, 1200
Nutrition: Need for better food, 334
Old age: Hospital admission for the senile, 874
Pensions increases, 874
Pneumoconiosis and Byssinosis Benefit Scheme, 1952, 552
Poison antidotes on labels, 1413
Police: Sickness in the Metropolitan Police, 1035
Road safety; tests for blood alcohol and eyesight, 552
"Roter" tablets, 552
Royal Cancer Hospital; change of name suggested, 1360
Surgical boots, 497
Tea, the national drug, 552
Teething powders, 874
Telephones: Party lines for doctors, 1360
Tobacco smoking and cancer of lung, 335
Tropical disease: Facilities for treatment in Britain, 1360
Tuberculosis in foreign workers, 1412
— in Singapore, 1413
— Incidence in Tyneside, 609
— Sanatoria waiting-lists, 497
— Scotland, 334
— Tyneside, 609
Unpunctual doctors, 1412

PARNELL, R. W.: Debauchery of honest words, 924
Paronychia, chronic, treatment, 58
Parotitis, suppurative, acute, in newborn (R. H. R. White), 1232
PARSONS, Alfred Robert, obituary of, 549, 664

Parsons Memorial Lectureship, 281
Partnership: Doctor's action against ex-partners fails (medico-legal note), 1412
— restrictive covenant appeal dismissed (medico-legal note), 111
PARTRIDGE, A. B.: Period of gestation, 1192
PARTRIDGE, A. J.: Pulmonary embolism from calf veins, 1405
P.A.S. *See* p-Aminosalicylic acid
PASSE, E. R. G.: Stellate-ganglion block, 486
PATERSON, D. H., appointed Clinical Professor of Paediatrics, University of British Columbia, 225
PATEY, D. H.: Pilonidal sinus, 1356
Pathology: *Atlas der pathologischen Anatomie* (R. Rössle and K. Apitz), 908
— clinical: *Clinical Pathological Data* (C. J. Dickinson), 311
— — 48th meeting of Association of Clinical Pathologists, 1039
— International Conference of Geographical Pathology, 1364
Patients in hospital: silence of consultants, 1195, 1244
PATRICK, I. T.: Malaria, 491
PAULETT, J. D. *See* FRANKLIN, L. M., 485
PAULLEY, J. W.: Cough fracture in late pregnancy, 162
PAVEY, Agnes E.: *Story of the Growth of Nursing as an Art, a Vocation, and a Profession*, 3rd ed., 311
PEACE, Percy Clift, obituary of, 224
PEAKE, H. L.: Malaria, 765
PEARSON, Henry Carden, obituary of, 717
PEASE, J. C., and SALT, R.: Syringe gauge for insulin, 1129
PECK, S. M., and KLEIN, G.: *Therapy of Dermatologic Disorders, including a Guide to Diagnosis and a Dermatologic Pharmacopeia*, 1011
Peckham Health Centre as medico-sociological research unit, 317 (A); 490 (C)
PEEL, J. *See* OAKLEY, W., 1248
PEILL, S. G.: What doctors prescribe, 545
PEMBERTON, J.: Invite Russian doctors, 165
PEMBREY, R. E. J.: Vomiting during anaesthesia, 711
Pemphigus neonatorum, notification of, 448, 614
PENFOLD, J. B.: Lipoma of the hypopharynx, 1286

PENICILLIN:
Accumulation of diethylaminoethanol ester of penicillin in inflamed lung tissue (J. Ungar and P. W. Muggleton), 1211 (O); 1238 (A)
Discoloration in syringes, 1205
In the lungs, 1238 (A)
Penicillin cream dangerous, 545, 713, 817, 924
Pulmonary aspergillosis following administration, 523
Therapy in common cold, 385
— in pneumonia, 37 (A)
— in tetanus neonatorum, 470

Penis: Neonatal priapism, 614
PENMAN, A. C. *See* EDWARDS, P. W., 1224
PENMAN, John, obituary of, 1305
Pensions and insurance, 207 (L)
— increases (parliamentary note), 874
PENTONY, John Joseph Kevin, obituary of, 1198

PEPTIC ULCER:
Childhood factors in duodenal ulcer, 109
Gastric carcinoma and acute perforation, 109, 164, 330
Peptic Ulcer: Clinical Aspects, Diagnosis, Management (Editor: D. J. Sandweiss), 1233
Relationship to carcinoma, 288
Simple gastric ulcer and carcinoma, 221
Treatment with "robaden" and enterogastrone, 109

Periodicals. *See* Journals
Persian Gulf Medical Society, 114
Perthes's disease, prognosis in, 1311
PETCH, C. P.: Carcinoma of the stomach, 492, 1029
PETERS, R. A., knighthood conferred on, 44
Pethidine hypersensitivity and histamine, 324
Petrol tax, 644 (A)
Phaeochromocytoma with negative benzodioxane test (H. P. Tulloh), 531 (O)
PHANSOMBOON, S., and POLLAK, O. J.: Study on the A factor as a cause of haemolytic disease in premature babies, 1170 (O)
Pharmacy: Association of British Pharmaceutical Industry, 982
Pharynx: Cancer of hypopharynx and its surgical treatment (R. W. Raven), 951 (O); 961 (L); 1358 (C)
— *Esophagus and Pharynx in Action. A Study of Structure in Relation to Function* (W. Lerche), 639
PHEMISTER, Dallas Burton, obituary of, 166
Phenobarbitone eruptions, severe and fatal (I. B. Sneddon and A. W. D. Leishman), 1276; 1405 (C)
PHILIPP, E. E.: Isonicotinic hydrazide, 1028
— and SKELTON, M. O.: Congenital diaphragmatic hernia in siblings, 1283 (O)
PHILLIPS, A.: Social trends and home confinements, 1353
PHILLIPS, A. F.: Period of gestation, 1354
PHILLIPS, John, obituary of, 225
PHILPOTT, L. A.: Danger of extracting many teeth at once, 222
PHILPOTT, M. G.: Gangrene of unknown aetiology in the foot, 201
Photography: Exhibition of medical illustration at B.M.A. House, 1293, 1363
— *Photography of Patients* (H. L. Gibson), 1394
Photosensitivity: Relation between sensitivity to thiopentone, sulphonamides, and sunlight (F. Evans and J. Gould), 417 (O); 477 (A)
— and porphyrians, 477 (A)
Physician. *See* Medical practitioner
Physiology: *Human Physiology* (C. C. Chatterjee), 857
— *Human Physiology* (B. A. Houssay and others), 585
— *Textbook of General Physiology* (H. Davson), 364
Physiotherapy: Re-education in walking, 1300
— *Treatment by Manipulation*, 2nd ed. fully revised (H. J. Burrows and W. D. Coltart), 1233
PICK, M. P.: Night pain with fractures, 274
PICKERILL, H. P.: Skin banks for wartime, 1194
— Limitations of A.C.T.H., 380
PICKERING, G. W.: Allergy and A.C.T.H., 1207 (O)
PICKETT, K. G. *See* CLEMENTS, E. M. B., 1300
Picture Post and the treatment of cancer: report of investigating committee, 1236 (L); 1256 (parliamentary note)
Pigmentation: Incontinentia pigmenti; report of case, with summary of 29 cases from the literature (A. W. Franklin), 75 (O); (correspondence), 277, 439
Pilonidal sinus of axilla (I. Aird), 902 (O); 1356 (C)
PIM, D. C., and OUTHWAITE, J. E.: Coronary disease in doctors, 651
PINEY, A.: Stellate-ganglion block, 486
— Multiple myelomatosis, 558
— *Handbook of Diseases of the Blood*, 423
Pink disease. *See* Acrodynia
PIRIE, N. W. *See* CLARK, F. leG., 856
PIRQUET, C. VON, and SCHICK, B.: *Serum Sickness*, 1118
Pitocinase activity of plasma during pregnancy, 622

PITUITARY:
Anterior hormones in pregnancy, 621
Hypopituitarism; A.C.T.H. in treatment (C. F. Rolland and J. D. Matthews), 1220 (O)
— anaemia of (V. K. Summers), 787 (O)
— effect of A.C.T.H. on capillary resistance in, 994
— treatment of hypopituitary coma (H. L. Sheehan and V. K. Summers), 1214 (O)
— with pituitary tumours; cortisone in treatment of stupor associated with hypopituitarism (J. E. Caughey, A. James, and E. K. MacLeod), 1216 (O)
Irradiation for diabetes, 1418
Posterior hormones in pregnancy, 622

Placebos: their usefulness in therapeutics, 149 (L); 326 (C)
Plasmapheresis: use in blood donors (J. A. Grifols-Lucas), 854
Plasmodium vivax, proguanil-resistance in Malayan strains of (T. Wilson, D. S. Munro, and D. R. Richard), 564 (O); 590 (A)
Plastic surgery. *See* Surgery, plastic
Plastics in surgery, 750 (L)
— *see also* Acrylics
PLATT, R.: Mercury in paediatrics, 484
— Structural and functional adaptation in renal failure, 1313 (O), 1372 (O); 1396 (L)
PLAYER, H. P. *See* LOCKET, S., 254
Pleural effusion and pulmonary consolidation associated with acute polyarthritis (A. W. Lees), 246 (O); (correspondence), 765
— — tuberculous, primary, in older age groups (R. F. Robertson), 133 (O)
Pleurodynia, epidemic. *See* Bornholm disease
Pneumoconiosis: Benefits for sufferers, 1089
— in gold mines, 965 (A)
Pneumoconiosis and Byssinosis Benefit Scheme, 1952 (parliamentary note), 552
Pneumoencephalography; value in diagnosis of fits (J. Marshall and C. W. M. Whitty), 847 (O); 1194 (C)
Pneumonectomy, first successful performance of, 986
— for bronchiectasis; indications and contra-indications, 557

PNEUMONIA:
Apical, meningeal signs in, 829
Atypical, primary, 110
Clinical features of the pneumonias undergoing virus tests (G. H. Jennings), 123 (O)
Effect of "terramycin," 419
Graphs (vital statistics), 54, 114, 170, 226, 282, 336, 392, 445, 499, 553, 610, 666, 721, 774, 827, 876, 928
Recent history; Osler Club symposium, 156
Treatment, choice of antibiotic, 37 (A)

Pneumothorax, spontaneous, from secondary sarcoma of lung, 713
POAD, W. H. P.: Humid climate and respiratory diseases, 104
Poisons Rules: Dangerous drugs in the anaesthetic room; legal and practical requirements for recording, 1365
Police: Sickness in the Metropolitan Police (parliamentary notes), 1035

POLIOMYELITIS:
Acute poliomyelitis, and polioencephalitis; graphs (vital statistics), 170, 981, 1038, 1088, 1140, 1203, 1256, 1308, 1362, 1415; notifications, 1255
And They Shall Walk. The Autobiography of Sister Elizabeth Kenny, 802; 1082 (C)
Gift for research, 877
Immunization, 270
In mother and newborn infant (G. A. Wright and T. K. Owen), 800 (O)
In 1948–51, 391

POLIOMYELITIS (*continued*):
Infantile Paralysis Fellowship; Edinburgh Group, 744
Outbreak of encephalitis possibly due to poliomyelitis virus (A. M. Barrett, D. Gairdner, and A. M. McFarlan), 1317 (O)
Returns of paralytic and non-paralytic cases, 483
Royal College of Physicians Committee, 1131
Treatment, early (G. P. Mitchell), 649; 923 (C)
— of trophic ulcers, 1312

POLLAK, O. J. *See* PHANSOMBOON, S., 1170
POLLARD, Walter Henry, obituary of, 389
POLUNIN, I.: Epidemiology of hypertension, 1190
Polyarthritis, acute, with pulmonary consolidation and pleural effusion (A. W. Lees), 246 (O); (correspondence), 765
"Polycentron" vaccinator, 440
Polycythaemia vera, splenogram in, 989
Polyoxyethylene ethers; effect on tuberculin sensitivity, 680; 702 (A)
Polyserositis complicated by acute beriberi; case in Britain (J. Thomson and K. R. Keay), 295 (O)
POOLE, J. C. F., and SUMMERS, G. A. C.: Correction of E.S.R. in anaemia; experimental study based on interchange of cells and plasma between normal and anaemic subjects, 353 (O); 369 (A)
POOLE, L. T.: Gadgets for the armless, 778
POPPEL, M. H.: *Roentgen Manifestations of Pancreatic Disease*, 960

POPULATION:
Census changes in 1951, 226
Demographic revolution, 1289 (L)
Distribution changes shown by 1951 census (vital statistics), 283
Human Fertility; The Modern Dilemma (R. C. Cook), 145
The Malthusian Controversy (K. Smith), 907; 1134 (C)

Porphyrins, photosensitivity to, 477 (A)
POSNER, E.: Shortage of *x*-ray films, 274
POTI, S. J. *See* CHAUDHURI, R. N., 568
POTTINGER, D.: Wooden heel for walking plasters, 710
POWELL, Mary: *Orthopaedic Nursing*, 696
POYNTER, F. N. L., and BISHOP, W. J.: *A Seventeenth Century Doctor and his Patients; John Symcotts, 1592?–1662*, Bedfordshire Historical Record Society Publications, Vol. XXXI (Nova et Vetera) (C. Singer), 1350
Prayers for Use in Hospitals, 534

PREGNANCY: *See also* Insemination, artificial; Labour; Obstetrics
Addison's disease (T. A. Richards), 421; 708 (C)
Anaemia of, 476
— "physiological": effect of iron therapy (Nancy Benstead and G. W. Theobald), 407 (O); 476 (A); 599 (C), 817 (C)
Cough fracture, 162, 274
Diabetes mellitus in, conduct of (H. H. F. Barns and M. E. Morgans), 1058 (O); 1248 (C)
Diagnosis; accuracy of various tests compared, 1366
Diagnostic tests, 501; 724 (C)
Duration of gestation (T. McKeown and J. R. Gibson), 938 (O); 1132 (C), 1192 (C), 1354 (C); statement to Royal Commission on Marriage and Divorce, 1244
Exercises Before Childbirth (Kathleen Vaughan), 1118
Full-time pregnancy associated with hydatidiform mole (Elizabeth M. Rose), 1285
Herpes gestationis, 230
Hormone assays in normal and abnormal pregnancies (G. I. M. Swyer), 619 (O)
Medical Disorders During Pregnancy (Editors: S. Clayton and S. Oram), 1010
Megaloblastic anaemias of pregnancy; response to animal protein factor; preliminary report (H. Foy, A. Kondi, and A. Hargreaves), 852 (O)
Naevus, 162
Paroxysmal tachycardia, 174
Progesterone pellet implantation; prophylactic value in habitual abortion (P. M. F. Bishop and N. A. Richards), 244 (O); 428 (L); (correspondence), 437, 440, 604, 819, 923
Preparation for "natural childbirth," 1302
Pseudocyesis, 538 (A); 657 (C), 816 (C)
Pulmonary tuberculosis and pregnancy (J. R. Edge), 845 (O); 862 (A); (correspondence), 1030
Termination in mitral stenosis, 1052
Torsion of Fallopian tube (N. P. Ellison and J. S. W. Chambers), 694; 1251 (C)
Toxaemia, diet in, 723
— hormonal findings in, 622
Tubal, interstitial; case (J. S. Leverton), 421

Pregnanediol assay in pregnancy, 620
Pregnenolone in arthritis, 1206

PREPARATIONS AND APPLIANCES:
Aid to analgesia of larynx and trachea (T. Dinsdale), 707
Cannulae, plastic, introducer for (J. V. Mitchell), 435
Hydrostatic bag for dilatation of the cardia (H. Burge), 1245
Insulin, syringe gauge for (J. C. Pease and R. Salt), 1129
Laryngoscope blade for infants (R. Bryce-Smith), 217

PREPARATIONS AND APPLIANCES (*continued*):
Mucus extractor for use on the newborn infant (H. H. Fouracre), 598
Operating glasses (G. Romanes), 1351
Rotatable sheath resectoscope (J. E. Semple), 46
Simple pulse indicator (V. J. Keating), 1188
Prescribing: *National Formulary*, 1952, 701 (A); 924 (C)
— Prescription charges in N.H.S., 283, 665, 719, 772; 1181 (A)
— Survey of 17,301 prescriptions on Form E.C. 10 (D. M. Dunlop), 292 (O); 312 (L); (correspondence), 435, 484, 545, 599, 709
Press, lay; doctors' opinions in, 599
PRESTON, T. W.: Shortage of *x*-ray films, 327
— Medical treatment of hypertension, 709
PRETTEJOHN, E.: Local aureomycin for aphthous stomatitis, 1082
Preventive medicine in relation to curative medicine, 327
Priapism, neonatal, 614
PRICE, J. M.: Pre- and post-operative treatment of dehydration, 48
— and ROGERS, W. J. B.: Succinylcholine as relaxant in electro-shock, 1027
Prickly heat, treatment by increased salt intake, 819
Printing costs, 629
"Priscol" for relief of acute left ventricular failure (D. Wheatley), 1174 (O)
— in cerebral arteriosclerosis, 98 (A); 106 (C)
Prison medical officers; annual conference, 1309
PRITCHARD, J. J., appointed Professor of Anatomy, Queen's University, Belfast, 1307
Prizes: Begley Prize, 875
— Conway Evans Prizes, 335
— Hallett Prize, 443
— Stewart Prize, 1184 (A)
— "Ulisse" Prize for 1952, 612
Procaine, intravenous, for pain relief, 266 (A)
Proctalgia fugax; aetiology and treatment, 669, 710, 880
Proctology: Brazilian Congress, 722
Professional prospects; comparison of the position of doctors with that of other professional people (R. Lewis), 615 (O)
Progesterone assay in pregnancy, 620
— pellet implantation; prophylactic value in habitual abortion (P. M. F. Bishop and N. A. Richards), 244 (O); 428 (L); (correspondence), 437, 440, 604, 819, 923

PROGUANIL:
Absorption and excretion, 570
Chemotherapeutic effect, 568
Intramuscular, 571
Parenteral, 570
Proguanil-pamaquin therapy in malaria, 571
Resistance to, 570
— in Malayan strains of *Plasmodium vivax* (T. Wilson, D. S. Munro, and D. R. Richard), 564 (O); 590 (A)
Toxicity, 570

Prostatectomy; effect on fertility, 670
— late results of perurethral resection (T. L. Chapman and J. W. Sutherland), 72 (O)
— resectoscope with rotatable sheath (J. E. Semple), 46
Prostatic abscess; case (E. Dunlop and J. Hosford), 1392
— obstruction, and inguinal hernia; a new approach (T. Moore), 362; 547 (C), 605 (C)
Protein sensitization, tissue reaction to (J. R. Squire), 1 (O); 35 (L); 165 (C)
Pseudocyesis, 538 (A); 657 (C), 816 (C)
Psittacosis: ban of importation of parrots, 27

PSYCHIATRY:
Carbon Dioxide Therapy. A Neurophysiological Treatment of Nervous Disorders (L. J. Meduna), 1072
Mental disease a symptom of physical defect, 972
Méthodes Biologiques en Clinique Psychiatrique (J. Delay), 960
Psychiatric case and the law (A. Kennedy): 1. General principles, 754; 2. Admission to hospital, 810; (correspondence), 921, 971, 1081, 1082, 1193, 1303, 1407
Scottish Western Region Association of Psychiatrists, 228
Shock therapy with acetylcholine, 964 (A); 1082 (C)
Soviet Psychiatry (J. Wortis), 473

Psycho-analysis, Adlerian and Freudian compared, 447
— *Essays in Applied Psychoanalysis*, vol. 2 (E. Jones), 261
— *Psychoanalysis of Elation* (B. D. Lewin), 364
Psychology: *Inferiority Feelings in the Individual and the Group* (O. Brachfeld), 585
— Jung's contributions, 314 (L); 655 (C)
Pteroylglutamic acid added to iron pills, 599
— — in megaloblastic anaemia of pregnancy, 1226
Public health: *The Making of our Towns* (Sir William Savage), 1177
— — *Principles of Public Health Administration* (J. J. Hanlon), 959
— — *Public Health is People* (Ethel L. Ginsburg), 857
— — *Rosenau's Preventive Medicine and Hygiene*, 7th ed. (K. F. Maxcy), 1338
Puerperal pyrexia and puerperal fever; graphs (vital statistics), 171
Pulse indicator, 1188
PURCHASE, W. B.: Attempted suicide, 761
Purpura and pancytopenia complicating arsenotherapy (M. G. Nelson), 300 (O); 714 (C)
— Schönlein-Henoch (anaphylactoid), treated with A.C.T.H. (E. J. S. Woolley), 259
— thrombocytopenic and amyloidosis in Hodgkin's disease (R. C. Gledhill and A. J. Shillitoe), 1336
— — and subsequent pregnancy, 932
— — effect of A.C.T.H. and cortisone, 1262
PURVIS, John Allen Spottiswoode, obituary of, 332
PURVIS, V.: Iridectomy in congestive glaucoma, 490
PYKE, D.: Family planning, 1354
— W.H.O. and birth control, 1405
PYKE, Margaret: Family planning, 1028
"Pyrotenex"; dermatitis after use (C. W. Ward), 905; 1249 (C)
Pyuria, effect of "terramycin" in, 420

Q

Quackery: Pendulum swinging in treatment of tuberculosis (medico-legal note), 665
Queen Elizabeth II, accession of, 367 (L)
Birthday Honours List, 1299
Honorary physicians to, 983
Loyal Address of B.M.A., 482
Patron of B.M.A., 1340
Quervain's disease, 646
Questions and Comments. *See* Any Questions?
Quinine, intramuscular, in treatment of malaria, 104, 220, 491
Quino-pamaquin therapy in malaria, 571

R

Rabies control, 931
— in Israel, 244
RACE, R. R., elected F.R.S., 703
RADDEN, H. G., appointed Professor of Dental Surgery, University of Manchester, 1307
Radio-isotopes for location of cerebral tumours, 428; 549 (C)

RADIOLOGY:
Dangers of *x*-ray mirror for dental fluoroscopy, 827
Disadvantages of small diagnostic units, 218
Faculty of Radiologists; Fellows elected, 281
International congress, 828
Lehrbuch der Röntgendiagnostik, 5th ed. (H. R. Schinz, W. E. Baensch, and others), 586; 1031 (C)
Roentgen Manifestations of Pancreatic Disease (M. H. Poppel), 960
Students' Radiological Mathematics; Elementary Mathematics Applied to Medical Radiology (L. A. W. Kemp), 908
X-ray appearances in asthma; 200 cases (H. Royle), 577 (O)
X-ray films, shortage of, 49, 94 (L); (correspondence), 107, 219, 274, 327, 385, 600
— — suggestions for economy in use, 219;
— — wastage of, 223

Radiotherapy: David Anderson-Berry Prize, 668
Radium: Terms for supply to N.H.S. hospitals, 827
Ramón y Cajal, S., centenary of birth of, 983
RAND, H. W.: *The Chordates*, 91
RANDALL, J. T. *See* BUTLER, J. A. V., 856
RAPER, Henry Stanley, appreciation of, 165; (for obituary, see *Journal*, 1951, **2**, 1527)
RATTENBURY, G. *See* FRANKLIN, A. W., 161
RAVEN, R. W.: Cancer of the hypopharynx and its surgical treatment, 951 (O); 961 (L); 1358 (C)
— and GOUGH-THOMAS, Joan: Educating the public about cancer, 381
RAVENHILL, Thomas Holmes, obituary of, 1032
Raynaud's disease; results of sympathetic ganglionectomy and pre-ganglionic section compared (J. B. Kinmonth and G. J. Hadfield), 1377 (O)
Rayon surgical dressings, 1361
READ, J.: *William Davidson of Aberdeen* (C. Singer) (Nova et Vetera), 380
Reading, effect on cataract, 651
Rectum, congenital absence of, treatment of, 558; 762 (C), 1084 (C)
— Proctalgia fugax, 669, 710, 880
REDFEARN, J. W. T.: Effect of fatigue on finger tremor, 816
REDMAN, T. F.: Intra-partum uterine rupture, 438
REES, Sir John Milsom, obituary of, 975, 1137
REES, R. J. W. *See* HART, P. D., 680
REESE, A. B.: *Tumors of the Eye*, 638
Refraction, Textbook of (E. F. Tait), 1117

REFRESHER COURSE FOR GENERAL PRACTITIONERS:
Abortion, medico-legal aspects of (D. Teare), 915; 911 (A)
Anaesthesia for minor procedures (W. W. Mushin), 431; 711 (C)
Atomic structure; basic principles (Sir James Walton), 99
Backache, clinical assessment of (J. G. Bonnin), 540
Blindness, curable, common causes of (J. Foster), 318; 490 (C), 651 (C), 762 (C), 1083 (C)
Burns, treatment of (J. P. Bull and D. M. Jackson), 1018, 1078
Empyema (T. H. Sellors), 704
Food-poisoning (Joan Taylor), 593
Fractures in the aged (R. H. Metcalfe), 1240 (correction, 1366)

REFRESHER COURSE (*continued*):
Gall-bladder, diseases of (H. C. Edwards), 1125
Gonorrhoea, treatment of (T. E. Osmond), 863
Heart block (G. Bourne), 966
Heat and sunlight, clinical effects of exposure to (B. G. Maegraith); 1. Heat hyperpyrexia and thermogenic anhidrosis, 1345; 2. Heat exhaustion and sunburn, 1402
Immunization, pros and cons (R. Cruickshank), 212, 268; (correspondence), 385, 714
Micturition, frequency of (H. P. Winsbury-White), 372; 547 (C)
Parkinsonism (H. G. Garland), 153, 382, 490
Psychiatric case and the law (A. Kennedy): 1. General principles, 754; 2. Admission to hospital, 810; (correspondence), 921, 971, 1081, 1082, 1193, 1303, 1407
Rheumatoid arthritis (W. S. C. Copeman), 1185
Serum reactions and serum sensitivity tests (L. J. M. Laurent and H. J. Parish), 1294
Shoulder, painful (J. W. Withers), 40
Tenosynovitis and tendovaginitis (D. Ll. Griffiths), 645; (correspondence), 766
Warts of the feet (L. Forman), 480; 712 (C)

Regional hospital boards (parliamentary note), 770
Registrar-general: *Quarterly Return for Eire* (vital statistics), 226, 1256
— *Quarterly Return for England and Wales* (vital statistics), 982
— *Quarterly Return for Scotland*, 875
Rehabilitation: British methods applied in Austria, 555
— Government report on, 1039
— Yugoslav study team, 283
REID, Henry Charles Cecil, obituary of, 871
Reiter's disease; treatment with chloramphenicol, 1358
Reminiscences: *Just a Medley of Amusing Tales and Interesting Medical and Legal Experiences*, 365
Remploy Ltd., 1039
RENSHAW, Graham, obituary of, 389

RESEARCH, MEDICAL: *See also* Medical Research Council
Clinical research in the National Health Service (Sir Cecil Wakeley), 917; 974 (C), 1030 (C), 1196 (C)
Fellowships in clinical research offered by Medical Research Council, 56
French scholarships in medical science, 227
South African medical research fellowship, 227

Resectoscope with rotatable sheath (J. E. Semple), 46
Resins for peptic ulcer: Treatment with anion resins, 829
Respiration, diffusion, 430 (A); 1358 (C)
— Simple standard exercise test and its use for measuring exertion dyspnoea (P. Hugh-Jones), 65 (O); 95 (A)
Respiratory diseases: harmful effects of warm humid climate, 104
— infections, effect of "terramycin" in, 419
Reticuloses associated with haemolytic anaemia (D. R. C. Willcox), 1322 (O)
Retina, detached, treatment of, 1041
Retinitis pigmentosa, prognosis in, 118
Retirement in industry, 1409
Retrolental fibroplasia; problem of prematurity (B. A. Bembridge and others), 675 (O); 698 (L); (correspondence), 924, 1029
Review of Medical and Veterinary Mycology, 1400 (A)
REY, J.: *The Essays of Jean Rey. A facsimile reprint of the original edition of* 1610, *with an introduction by Douglas McKie* (C. Singer) (Nova et Vetera), 1188
Rh factor, 108
— — *Rh Blood Groups and their Clinical Effects*, 667; 870 (C)
— — verbal usage of CDE notation for Rh blood groups, 851

RHEUMATISM:
Agglutination reaction, 1153
Anomaly of sedimentation rate in rheumatic diseases (Sylvia Goldberg, L. E. Glynn, and E. G. L. Bywaters), 202
Cortisone and A.C.T.H., 1155
— in rheumatic carditis: Some preliminary observations (E. N. Chamberlain, J. D. Hay, and D. M. Freeman), 1145 (O); 1179 (L)
Empire Rheumatism Council; fifteenth annual meeting, 1024
Relationship between acute rheumatic fever, rheumatoid arthritis, and other conditions, 765
Rheumatic diseases in the U.S.A. (J. J. R. Duthie), 341 (O); 602
Some concepts of rheumatic disease (J. H. Kellgren), 1093 (O), 1152 (O)
Treatment with gamma rays, 1311

Rheumatoid arthritis. *See* Arthritis, rheumatoid
Rhinitis, allergic, 380
Rib: Cough fracture, 220
— — — in late pregnancy, 162, 274
Rice diet in hypertension, 587, 597
RICHARD, D. R. *See* WILSON, T., 564
RICHARDS, H., and YOUNGMAN, H. R.: Ultra-short-acting relaxants, 1334 (O); 1342 (A)
RICHARDS, H. J.: Dupuytren's contracture treated with vitamin E, 1328 (O)
RICHARDS, N. A. *See* BISHOP, P. M. F., 244, 604
RICHARDS, T. A.: Addison's disease and pregnancy, 421; 708 (C)

RICHARDSON, I. M.: Elderly workers in heavy industry, 815
RICHES, E. W.: Insulated diathermy haemostat, 1356
RICKARDS, A. G.: Megaloblastic anaemia of infancy, 1226 (O)
RIGGALL, F.: Treatment of cancer, 1029
"Rimifon," 858
Ringworm: Tinea capitis (*M. audouini*) in residential school (A. L. Walby), 1114 (O)
RINKEL, Lambert Ronald Joslin, obituary of, 51
RITCHIE, Jean R. S.: *Teaching Better Nutrition*, 145; 384 (C)
RIVLIN, S.: *Treatment of Varicose Veins and their Complications*, 260
ROACH, Elizabeth S. *See* KAREL, L., 1290
Road accidents (vital statistics), 282, 499, 721, 1363
— — in relation to alcohol, 47, 1413
— — motor-cycle accidents, 1183 (A)
— — relation to poor vision in drivers, 108
— safety, medical aspects of, 489
— — tests for blood alcohol and eyesight (parliamentary note), 552
"Robaden" and enterogastrone in peptic ulcer, 109
ROBBINS, Frank Hubert, obituary of, 495
ROBERTS, Ff.: Coal and cortisone; man-power in a sick society, 321; 437 (C)
— *The Cost of Health*, 642; (correspondence), 818
— Barrier of silence, 1195
ROBERTS, J. A. F.: Statures of university students and parents, 1133
ROBERTS, J. M.: Traumatic autografting, 274
ROBERTSON, J. D., and KIRKPATRICK, H. F. W.: Changes in basal metabolism, serum-protein-bound iodine, and cholesterol during treatment of hypothyroidism with oral thyroid and L-thyroxine sodium, 624 (O)
ROBERTSON, K.: Future use of cortisone and A.C.T.H. in general practice, 103
ROBERTSON, R. F.: Primary tuberculous pleural effusion in older age groups, 133 (O)
ROBINSON, Henry James, hundredth birthday of, 1141
ROBSON, H. N., and DUTHIE, J. J. R.: Further observations on capillary resistance and adrenocortical activity, 994 (O)
ROBSON, J. M. *See* KNOX, R., 1081
ROCHA E SILVA, M.: Concerning the mechanism of anaphylaxis and allergy, 779 (O); 807 (A)
Rochdale: smallpox in (vital statistics), 444, 498, 478 (A), 553, 610, 666, 720, 775, 875, 928, 981, 1038, 1088
ROCHE, A. E.: Hour-glass bladder, 583
ROCHFORD, J., and ROUALLE, H.: Xylocaine, 714
Rockefeller travelling fellowships in medicine, 445
RÖSSLE, R., and APITZ, K.: *Atlas der pathologischen Anatomie*, 908
ROGERS, A. T.: Future use of cortisone and A.C.T.H. in general practice, 102
ROGERS, K. B.: Effect of hypochromasia on the specific gravity and sedimentation of red cells, 1109 (O); (correction, 1418); 1407 (C)
ROGERS, W. J. B. *See* PRICE, J. M., 1027
ROITH, A.: Geographical tongue, 670
ROLLAND, C. F.: Diabetes mellitus in surgical patients, 737 (O)
— and MATTHEWS, J. D.: A.C.T.H. in hypopituitarism, 1220 (O)
ROMANES, G.: Operating glasses, 1351
ROMANOWSKI, E.: Herpes ophthalmicus and Parkinson's syndrome, 820
ROPER, M.: Proctalgia fugax, 880
ROSE, Elizabeth M.: Full-time pregnancy associated with hydatidiform mole, 1285
ROSE, L.: Technique of shock therapy, 330
— Prolonged respiratory paralysis after succinylcholine, 1027
ROSE, L. M.: Congenital defects in mongols, 276
Rosenau's Preventive Medicine and Hygiene, 7th ed. (K. F. Maxcy), 1338
ROSENHEIM, M. L.: Treatment of hypertension, 597; 587 (L)
ROSS, A. O.: Treatment of lightning pains, 1312
Ross Institute Industrial Advisory Committee, 479 (A)
"Roter" tablets (parliamentary note), 552
ROTH, Grace M.: *Tobacco and the Cardiovascular System. The Effects of Smoking and of Nicotine on Normal Persons*, 803
ROUALLE, H. *See* ROCHFORD, 714
ROUSSAK, N. J.: Fatal hypokalaemic alkalosis with tetany during liquorice and P.A.S. therapy, 360 (O)
ROWLEY, H. A. *See* DEWHURST, C. J., 973
Royal Beatson Memorial Hospital, 983
Royal Cancer Hospital; change of name suggested (parliamentary note), 1360

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS:
Appointments, 1307
Diplomas granted, 1036
Fellows elected, 443, 1307
Leverhulme research scholarships, 1415
Members admitted, 443, 1307

ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH:
Conjoint diplomas granted, 666
Fellows elected, 554, 1201
Members elected, 554
Officers elected, 281

ROYAL COLLEGE OF PHYSICIANS OF IRELAND:
Examination results, 53
Licences granted, 825
Members admitted, 391, 825

ROYAL COLLEGE OF PHYSICIANS OF LONDON:
Appointments, 335
Brain, Sir Russell, re-elected President, 862
Committee on poliomyelitis, 1131
Conway Evans prizes, 335
Diplomas awarded, 168, 336, 443, 875
Goulstonian Lectures, 1093, 1152, 1260 (correction)
Licences conferred, 335
Lumleian Lectures, 1313, 1372
Members elected, 335

ROYAL COLLEGE OF SURGEONS OF EDINBURGH:
Conjoint diplomas granted, 666
Fellows admitted, 112, 1307

ROYAL COLLEGE OF SURGEONS OF ENGLAND:
Appointments, 443, 1254
Arris and Gale Lecture, 59
Bland-Sutton bequest, 875
Causey, G., appointed Sir William Collins Professor of Anatomy, 168
Diplomas awarded, 168, 336, 443, 875, 1254
Faculty of Anaesthetists, 168, 825
Fellows elected, 665, 875, 1254
Hallett Prize, 443
Hospitals recognized 168, 665, 875, 1254
Licences conferred, 335
Lister Oration, 877
Members elected, 443, 665
Prizes and scholarships, 665, 1254
Royal College a "charity" (medico-legal note), 979
Thomas Vicary Lecture, 1254

ROYAL COLLEGE OF SURGEONS IN IRELAND:
Examination results, 53
Licences granted, 825

Royal Commission on Marriage and Divorce; evidence submitted by B.M.A., 1237 (L); 1242
Royal Dental Hospital and London School of Dental Surgery, 1115

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW:
Biennial dinner, 56
Conjoint diplomas granted, 666

Royal Household: Appointments, 116, 332
Royal Institute of Public Health and Hygiene: Harben Medal, 1142
Royal Sanitary Institute: Health Congress, 1021; 1131 (C), 1135 (C), 1196 (C), 1246 (C)
Health Week (1953), 1102

ROYAL SOCIETY:
Appointments, 53
Conversazione, 1182 (A)
New Fellows, 703 (A)

ROYAL SOCIETY OF MEDICINE:
Section of Epidemiology and State Medicine; Practical epidemiology, 483
Spread of infection in the family, 968
Section of General Practice; Child Management, 1189
Possible future use of cortisone and A.C.T.H. in general practice, 102 (correction, 230)
Section of Medicine; Treatment of hypertension, 597, 587; 587 (L)
Section of Obstetrics and Gynaecology; Present conception of trial labour, 1025
Section of Pathology; Drug resistance of micro-organisms, 434
Section of Physical Medicine; Conservative treatment of disk syndrome, 217
Section of Psychiatry; Attempted suicide, 1130
Different approaches to the problem of criminal responsibility, 45

Royal Statistical Society, prize, 500; study circle for examining statistical problems in medicine, 1040
ROYES, K. C.: Malaria, 491
ROYLE, H.: *X*-ray appearances in asthma; a study of 200 cases, 577 (O)
Rubella immunization, 270
— in pregnancy, as indication for therapeutic abortion, 915
RUSSELL, Albert Victor John Charles, obituary of, 389
RUSSELL, H. S.: Laryngeal epilepsy, 653, 1250
RUSSELL, P. F.: *Malaria; Basic Principles Briefly Stated*, 1395
Russia. *See* U.S.S.R.

S

SADLER, Agnes R. S.: Family planning, 868, 1132
St. Andrews University. *See* Universities: St. Andrews
St. John of Jerusalem, Order of; promotions and appointments, 160, (correction, 228, 340)
St. Thomas's Hospital; celebration of quatercentenary of refounding, 55
SALFIELD, D. J.: Archetype and schema, 655
Salivary tumour of hard palate, 600
Salmonella, food-poisoning due to, 593
— *dublin*; persistent urinary carrier with bilharziasis (W. S. Miller), 628 (O)
— *minnesota* infection, 38 (A); 53 (vital statistics)
— *montevideo* infection of secondary carcinoma, 1116
— *typhi*, absolute viability in water (J. F. D. Shrewsbury and G. J. Barson), 954 (O)
SALT, R. *See* PEASE, J. C., 1129
Salt intake, excessive, as cause of recurrent axillary boils, 174
SAMUEL, H. S.: Anaemia in pregnancy, 817
SAND, R.: *Advance to Social Medicine*, 1394
SANDERS, C. D.: Forgotten tourniquet, 605
SANDIFORD, B. R. *See* CARPENTER, K. Patricia, 142
SANDIFORD, H. A.: Hospitality for overseas visitors, 1246
SANDREY, J.: Prostatic obstruction and inguinal hernia, 547
SANDWEISS, D. J. (Editor): *Peptic Ulcer; Clinical Aspects, Diagnosis, Management*, 1233
SANGSTER, A. H.: Rapid cure of actinomycosis, 277
Sanitary inspectors' working party, 1089
Sarcoidosis of Boeck, calciferol therapy in, 58
— splenogram in, 989
SAUTTER, H.: *Trübungsforme der menschlichen Linse*, 908
SAVAGE, O. *See* COPEMAN, W. S. C., 397
SAVAGE, Sir William: *The Making of our Towns*, 1177
SAXTON, R. S.: Coal and cortisone, 437
— Sulphamerazine still dangerous, 870
SAYERS, W. J. H.: Sexual disorders in the female, 1135, 1247
Scalp, sebaceous cysts, removal of, 777
Scarlet fever, epidemiology, 317 (A)
— — graphs (vital statistics), 54, 114, 171, 226, 282, 336, 392, 666, 721, 775, 827, 876, 928
— — immunization, 270
Scarpa, Antonio, bicentenary of birth of, 1066
SCHACHTER, M.: Pethidine hypersensitivity and histamine, 324
SCHEINKER, I. M.: *Medical Neuropathology*, 639
SCHINZ, H. R., BAENSCH, W. E., and others: *Lehrbuch der Röntgendiagnostik*, 5th ed., 586; 1031 (C)
Schistosomiasis. *See* Bilharziasis
Schizophrenia: *Dementia Praecox or the Group of Schizophrenias* (E. Bleuler), 32, 326
— insulin dosage in coma therapy, 501
— physical characteristics associated with, 558, 932
SCHMASSMANN, H.: "Robaden" and enterogastrone, 109
SCHMID, J.: *Die Blutgerinnung in Theorie und Praxis*, 92
SCHMIDT-VOIGT, J.: *Herzschalldiagnostik in Klinik und Praxis*, 1233
SCHNEIDER, R. A. *See* DOUST, J. W. L., 449
Schönlein-Henoch (anaphylactoid) purpura treated with A.C.T.H., 259
School Medical Service, 210 (A); 490 (C); 1022
School of Pharmacy; granted Royal Charter of Incorporation, 1363
SCHORSTEIN, J.: Ethics of prefrontal leucotomy, 919
Sciatica, surgical treatment, 325
Science: *Common Sense of Science* (J. Bronowski), 653
— Overseas Science Students' Association, 929
Scientists, Chambers's Dictionary of (A. V. Howard), 310
Scleritis, aetiology of, 1417
Scleromalacia perforans, treated with streptomycin, 122
Sclerosis, disseminated; diagnosis, hereditary factors, and effect of pregnancy, 502
— — treatment with quaternary ammonium compounds, 880
"Scoline." *See* Succinylcholine
Scoliosis, "idiopathic," 219
Scorpion stings; treatment with emetine, 396, 614

SCOTLAND:
Causes of death in 1949 (vital statistics), 337
Height of Scotsmen, 1300
Registrar-General's Quarterly Return (vital statistics), 875
Reports of the Department of Health for Scotland and the Scottish Health Services Council, 1951
Tuberculosis, 334
— mortality in 1949 (vital statistics), 499

SCOTT, D. B.: Intra-partum uterine rupture, 973
SCOTT, J. A., appointed Medical Officer of Health and School Medical Officer of the London County Council, 446
SCOTT, L. G. *See* BRODRIBB, H. S., 1060
SCOTT, R. A. M.: Anticoagulants in leukaemia, 162
SCOTT, R. B.: Some medical aspects of tobacco-smoking, 671 (O); (correspondence), 869, 975
SCOTT, W. C. M.: Meyer and Bleuler, 326
Scottish Society of Anaesthetists, 1309
Scrotum, retention cysts of, 174
SCUTT, R.: Bullous lesions in leukaemia, 139 (O)
Seamen: *Medical Care of Seamen. Origin of Public Medical Service in the United States* (R. Straus), 33
Seasickness, prevention and treatment, 57, 1042
Sebaceous cysts of the scalp, removal of, 777
SEED, P. G.: Torsion of a Fallopian tube, 1176; 1407 (C)
SELLORS, T. H.: Empyema, 704
— and LIVINGSTONE, J. L. (Editors): *Modern Practice in Tuberculosis* (in 2 vols.), 747
— *See* LING, T. M., 756
SELYE, H.: *Annual Report on Stress*, 1288
Semen; methods of collection for fertility estimation, 218, 328
SEMON, H. C.: Eczema of the hand in a child, 1418
SEMPLE, J. E.: Rotatable sheath resectoscope, 46
Sensation: Congenital indifference to pain, 144
Serratus anterior, testing for weakness of (Cynthia M. Walker), 1393
Serum reactions and serum sensitivity tests (L. J. M. Laurent and H. J. Parish), 1294
— — precautions against, 483
Serum Sickness (C. von Pirquet and B. Schick), 1118

Services. *See* Armed Forces
Sex education, 866
— hormone treatment of over-sexed girls, 986
— organs, maldevelopment of, 340, 614
— *Patterns of Sexual Behavior* (C. S. Ford and F. A. Beach), 1338
Sexual abnormalities; diagnosis of intersex (D. I. Williams), 1264 (O); 1292 (A)
— disorders in the female, 104, 273, 437, 656, 1135, 1247
— intercourse; absence of sensation during orgasm, 1092
SHACKLE, J. W.: Westergren and Wintrobe methods compared, 277
SHACKMAN, R., and GRABER, I. G.: Reciprocal changes in blood flow of the limbs and rectum during general anaesthesia, 1284 (O); 1409 (C)
SHAPIRO, B. G.: Male infertility, 656
SHATTOCK, F. M.: Vascular spasm, 487
SHAW, Raymond Henry, obituary of, 551
SHEEHAN, H. L., and SUMMERS, V. K.: Treatment of hypopituitary coma, 1214 (O)
Sheffield University. *See* Universities: Sheffield
SHEPPARD, M. D.: Proctalgia fugax, 880
Sheriffs, medical, 668
SHERLOCK, Sheila (Editor): *Liver Disease*, 1233
SHERMAN, D. A.: Prolonged respiratory paralysis after succinylcholine, 1135
SHERMAN, H. C.: *The Nutritional Improvement of Life*, 145; 384 (C)
SHERRIFF, G. G.: Latin comes to life, 1304
SHERRINGTON, Sir Charles Scott, obituary of, 606; 589 (L)
SHERWOOD, Denise G. *See* SHERWOOD, P. H., 1406
SHERWOOD, P. H., and SHERWOOD, Denise G.: Responsibility and registration, 1406
SHIELDS, Sir Douglas Andrew, obituary of, 549
Shigella flexneri 103 Z, epidemiology of human case of bacillary dysentery due to infection by (K. Patricia Carpenter and B. R. Sandiford), 142 (O)
Shigellae, absolute viability in water, 954
SHILLITOE, A. J. *See* GLEDHILL, R. C., 1337
SHIPMAN, J. J. *See* FRANKLIN, R. H., 947
SHIRCORE, J. O.: Treatment of tropical ulcers, 764
— Infective gangrene of the mouth, 975
SHIRLAW, J. T.: Tissue reactions to protein sensitization, 165
SHOCK, N. W.: *Trends in Gerontology*, 638
SHORT, A. R.: Educating the public about cancer, 276
SHORT, D. S.: Survey of pulmonary embolism in a general hospital, 790 (O); 809 (A); 920 (C), 1303 (C), 1405 (C)
Shoulder, painful (J. W. Withers), 40
SHREWSBURY, J. F. D., and BARSON, G. J.: Note on the absolute viability in water of *Salm. typhi* and the dysentery bacilli, 954 (O)
SHUBSACHS, S.: What doctors prescribe, 437
SHUTE, E. V. *See* SHUTE, W. E., 276
SHUTE, W. E., and SHUTE, E. V.: Diabetic gangrene, 276 (correction, 493)
Shwartzman phenomenon, 781
Sickle-cell trait in Southern India (H. Lehmann and Marie Cutbush), 404 (O); 426 (L); (correspondence), 920
— — in Western Nigeria: Survey of 1,881 cases in the Yoruba (D. B. Jelliffe and J. Humphreys), 405 (O); 426 (L); (correspondence), 763, 920
Sickness incidence, now and in the future, 321
Silicosis, aluminium therapy in, 829, 1206
SILVERSTONE, E.: Trauma and fibrosarcoma in foot, 958
Silvester, Henry Robert, portrait of, 227; portrait traced, 393
SIMMONS, J. S., WHAYNE, T. F., ANDERSON, G. W., and HORACK, H. M.: *Global Epidemiology; A Geography of Disease and Sanitation*. Vol. 2: *Africa and the Adjacent Islands*, 1010
SIMPSON, R. E. H.: Mumps with involvement of lacrimal glands, 1250
SIMPSON, S. L.: Some recent advances in endocrinology, 725 (O)
SIMPSON, W.: Is Latin dead? 1084
SIMS, H. C.: Credit to Switzerland, 1031
Sims-Black Travelling Professor, 443
Singapore; annual report of Medical Department (vital statistics), 114
SINGER, C.: *William Davidson of Aberdeen. The first British Professor of Chemistry* by J. Read (Nova et Vetera), 380
— *Caelius Aurelianus; On Acute Diseases and on Chronic Diseases*, edited by I. E. Drabkin, 433 (Nova et Vetera)
— Pioneers of anaesthesia (review of *The English Pioneers of Anaesthesia (Beddoes, Davy, and Hickman)* by F. F. Cartwright (Nova et Vetera), 706
— *Apologie and Treatise of Ambroise Paré* (Editor: G. Keynes) (Nova et Vetera), 1129
— *The Essays of Jean Rey. A facsimile reprint of the original edition of* 1610, *with an introduction by Douglas McKie* (Nova et Vetera), 1188
— *A Seventeenth Century Doctor and his Patients; John Symcotts*, 1592?–1662 (F. N. L. Poynter and W. J. Bishop), Bedfordshire Historical Record Society Publications, Vol. XXXI (Nova et Vetera), 1350
— *Translation of Galen's Hygiene (De Sanitate Tuenda)* by R. M. Green (Nova et Vetera), 1404
SINGLETON, W. T.: Deterioration of performance in fatigue, 816
SKEELS, Percy Bernard, obituary of, 225
SKELTON, M. O. *See* PHILIPP, E. E., 1283
SKEVINGTON, Sir Joseph Oliver, obituary of, 660
SKIN: *See also* Dermatitis; Dermatology
Bullous lesions in leukaemia (R. Scutt), 139 (O)
Grafts; banks for wartime, 1194
Handbook of Diseases of the Skin (H. O. Mackey), 1071
Incontinentia pigmenti; report of case, with summary of 29 cases from the literature (A. W. Franklin), 75 (O); (correspondence), 277, 439
Lesions in ulcerative colitis, 1194
Marbled, treatment of, 396
M.R.C. research unit, 929
Non-urticarial dermatoses treated by antihistamines (P. Inman and I. C. Cowan), 1064 (O)
Reactions to protein substances, 2
Resistance to ultra-violet light, 162
Staining due to gold watch, 1042
Skull; radiological changes in dystrophia myotonica (J. E. Caughey), 137 (O)
SLATER, E., and WOODSIDE, Moya: *Patterns of Marriage. Study of marriage relationships in the urban working classes*, 91
Sleep, cause of anoxia in, 651
— Studies on the physiology of awareness; anoxia and the levels of sleep (J. W. L. Doust and R. A. Schneider), 449 (O); 475 (L); (correspondence), 971
Sleeping posture in phthisis, 316 (A)
SMALLPEICE, V., and COXON, M.: Congenital duodenal obstruction and mongolism, 384
— *See* BEMBRIDGE, B. A., 675
SMALLPOX:
Diagnosis of Smallpox, 204
In Hampshire (vital statistics), 775
In Lancashire (vital statistics), 444, 498, 478 (A), 553, 610, 666, 720, 775, 875, 928, 981, 1038, 1088
In Mexico (vital statistics), 1088
In Swansea (vital statistics), 981
Notifications (vital statistics), 826
Vaccination, 268
— case of accidental generalized vaccinia (O. A. Finn, J. C. Dick, and J. S. Stevenson), 1067 (O); (correspondence), 1134, 1249
— death following (vital statistics), 1037
— instrument for, 440
— reactions to, 483
Variola minor in Lancashire (vital statistics), 498, 478 (A), 553, 610, 666, 720, 875, 928, 981, 1038, 1088
SMITH, D. A.: Geographical tongue, 670
— *See* MCGREGOR, I. A., 730
SMITH, D. P.: Congestive and simple glaucoma, 869
SMITH, E. L.: Isolation and chemistry of vitamin B_{12}, 160
SMITH, Edward Davison, obituary of, 717
SMITH, Frederick Hugh, obituary of, 279
SMITH, Isabel G.: Skin lesions in ulcerative colitis, 1194
SMITH, James Adam, obituary of, 279
SMITH, K.: *The Malthusian Controversy*, 907; 1134 (C)
SMITH, P. S.: Foot and mouth, 1251
SMITH, Sheila: Rh in practice, 870
SMITH, Sir Sydney: Death on the roads, 47
Smoking. *See* Tobacco
SNAITH, L. *See* CAMPBELL, H., 1408
SNEDDON, I. B.: Local aureomycin for aphthous stomatitis, 1246
— and COLQUHOUN, J.: Granulomatous ulcer of nose treated with chloramphenicol, 298 (O); 491 (C)
— and LEISHMAN, A. W. D.: Severe and fatal phenobarbitone eruptions, 1276 (O); 1405 (C)
Sneezing; tale of three sneezes, 1358
SNELLGROVE, D. R.: What doctors prescribe, 485
SNIPER, W.: Methonium compounds and muscle relaxants, 492
Social medicine: *Advance to Social Medicine* (R. Sand), 1394
— services: *Handbook to the Social Services in London*, 283
— — *Social Services*, 207
Society for General Microbiology, 1090, 1204
— of Medical Officers of Health, Andrew Topping elected President, 1142
Society for Relief of Widows and Orphans of Medical Men, annual general meeting, 1415
See also
Chelsea Clinical Society
Devon and Exeter Medico-Chirurgical Society
Harveian Society of London
Heberden Society
Hunterian Society
Manchester Medical Society
Medical Society of London
North of England Obstetrical and Gynaecological Society
Nutrition Society
Ophthalmological Society of the United Kingdom
Royal Society
Royal Society of Medicine
Royal Statistical Society
Scottish Society of Anaesthetists
Ulster Paediatric Society
SOFFER, L. J.: *Diseases of the Endocrine Glands*, 748
SOLOMON, G.: Is Latin dead? 1197
SOLOMONS, B.: Male infertility, 218
SOLOMONS, B., jun.: Goat's milk and infantile eczema, 973
SOMNER, A. R.: Side-effects of dihydrostreptomycin, 1302
Songo fever, 477
Sonne dysentery, familial spread of, 969
SOPHIAN, J.: Vascular spasm, 382
SORSBY, A., UNGAR, J., and BAILEY, N. L.: Streptomycin in ophthalmology, 119 (O); 151 (A)
— *Genetics in Ophthalmology*, 203
— (Editor): *Systemic Ophthalmology*, 310
South Africa: Rift Valley fever, 267 (A)
South African medical research fellowship, 227
South-west Regional Paediatric club, 1415
SOWRY, G. S. C. *See* HEPTINSTALL, R. H., 525
Spain: Homes for doctors, 564
— Medical films, 568
SPALTON, L. M.: L-*Nor*adrenaline, 223
Spectacles: Operating glasses (G. Romanes), 1351
SPECTOR, W. G. *See* BUTLER, N. R., 1168
Speech: Disabilities in children, 36 (A); 325
Spens Committee and income tax, 913 (A)
— — and Mr. Bevan, 1291 (A)
Spermatozoa, viability in female genital tract, 285
SPERRY, W. L.: *Ethics of Medical Practice*, 1118
SPICER, C. C.: Sonne dysentery, 969
Spinal cord: Angioma venosum racemosum with angiomatous lesions of skin and omentum (I. Gilbert), 468 (O)
SPINE:
Hazards of manipulative treatment in limbo-sacral disk protrusions; case (R. Hafner), 361 (O); (correspondence), 655
"Idiopathic" scoliosis, 219
Intervertebral disk lesions; conservative treatment, 217
— — — sponge-rubber collar for, 972
Spondylitis, ankylosing; treatment with cortisone and A.C.T.H. (F. D. Hart), 188 (O)
Spirit, surgical, purification after use, 229
Spleen, leukaemic, spontaneous rupture of (G. H. Cooray), 693
— sarcoma of (P. Story and W. E. Gibb), 990 (O)
— spontaneous rupture of (J. A. V. Nicoll), 801
— traumatic rupture, incidence, 223
— wandering, during first decade of life (C. L. Hall), 957; 1407 (C)
Splenic puncture (J. B. Chatterjea, C. M. Arrau, and W. Dameshek), 987 (O); 1016 (A)
— — technique of, 987
Splenins A and B in rheumatoid arthritis, 18, 438, 603
Spondylitis, ankylosing; treatment with cortisone and A.C.T.H. (F. D. Hart), 188 (O)
SQUARE, William Holberton, obituary of, 111
Squint: *Surgery of the Oblique Muscles of the Eye* (W. H. Fink), 747
SQUIRE, J. R.: Tissue reactions to protein sensitization, 1 (O); 35 (L)
Stains from medicinal dyes, removal of, 1366
— on doctors' clothes, 173
STALLARD, B. E. W.: Incontinence in women, 277
STALLARD, John Prince, obituary of, 332
STAMM, T. T.: Sponge-rubber collar for cervical disk lesions, 972
STAMMERS, F. A. R.: Congenital dermal sinus and meningitis, 221
— Middle East tour, 500
STANDLEY, D. W.: Ethics of leucotomy, 1028
STANLEY, B. E. C.: Extensive resection of small intestine, 383
Staphylococcal septicaemia, fatal, following removal of tonsils and adenoids (E. J. G. Wallace and O. G. Jones), 1231
Staphylococcus albus, coagulase-positive, as cause of bacterial endocarditis (C. C. Cobb), 90
STATISTICS: *See also* Vital statistics
Applied Statistics, 1089
City of Birmingham Annual Abstract of Statistics, No. 1, 1931–1949 (Editor: R. Fadley), 908
Frances Wood Memorial Prize, 500
Probit Analysis, 1234
Role in regional hospital planning, with special reference to Birmingham Hospital Region (Vera Norris), 129 (O); (correspondence), 272, 437
Royal Statistical Society, study circle for examining statistical problems in medicine, 1040
Statures of a group of university students and of their parents (J. V. G. A. Durnin and J. B. deV. Weir), 1006 (O); (correspondence), 1133, 1300
Status asthmaticus; use of cortisone, 103
Steatorrhoea, "epidemic" abdominal colic associated with (M. E. M. Thomas), 691 (O)
— idiopathic; effect of cortisone (J. Badenoch), 356 (O)
— — occult, aetiological importance of, in megaloblastic anaemia of young adults (H. Conway), 1098 (O)
STEED, G. R.: Spontaneous pneumothorax from secondary sarcoma, 713
STEEL, S. J.: Acquired sensitivity to *para*-aminosalicylic acid, 415 (O)
Stellate ganglion block; indications and technique, 777
STENGEL, E.: Inquiries into attempted suicide, 1130
Sterility; treatment by tubo-uterine implantation (V. B. Green-Armytage), 1222 (O); 1355 (C)
Sterilization, sexual, during the puerperium, 1312
STEVEN, Peter Alexander, obituary of, 1199
STEVENSON, J. S. *See* FINN, O. A., 1067
STEWART, C. *See* MCNEISH, W. W. W., 744
STEWART, J. W. *See* COPEMAN, W. S. C., 397

Stewart, M. J., plaque and lectureship in honour of, 1416
— retirement fund, 1255
Stewart Prize, 1184 (A)
STOCK, William Stuart Vernon, obituary of, 1411

STOMACH:
Carcinoma (B. F. Swynnerton and S. C. Truelove), 287 (O); 313 (L); 492 (C), 708 (C), 1029 (C), 1301 (C)
— and acute perforation, 109, 164, 330
— associated conditions, 288
— perforation as sign of, 288
Stomach tube in trachea, 382, 486, 710, 715, 920
Surgery; peritonitis complicated by septic peritonitis following gastro-intestinal surgery (D. B. Irwin), 1379 (O)
Ulcer. *See* Peptic ulcer

Stomatitis, aphthous, treated with local aureomycin 1082, 1246
STORY, P., and GIBB, W. E.: Sarcoma of the spleen, 990 (O)
Strabismus. *See* Squint
Strangulation by Judo hold, 447
Strasbourg summer school, 338
STRAUS, R.: *Medical Care of Seamen. Origin of Public Medical Service in the United States*, 33

STREPTOMYCIN:
Dihydrostreptomycin, deafness due to, 1076
— deafness with use in tuberculous meningitis (J. T. Naismith), 796 (O)
— in tuberculosis; contraindications, 1076 (A)
— uncommon neurotoxic symptom associated with (P. O. Leggat and J. H. Gifford), 1008; 1302 (C)
In ophthalmology (A. Sorsby, J. Ungar, and N. L. Bailey), 119 (O); 151 (A)
Intra-articular injection in tuberculosis of knee (R. T. Ahern and G. P. Arden), 466 (O); (correspondence), 766, 974, 1194
Prevention of streptomycin resistance by combined chemotherapy; M.R.C. investigation, 1157 (O); 1179 (L); (correspondence), 1302
Pulmonary aspergillosis following administration, 523
Toxic effects, 1076 (A)
Treatment of pulmonary tuberculosis, 1224
— of tuberculous meningitis (S. R. Jamieson), 83 (O); (corrections, 165, 223); 329 (C)
— of pulmonary tuberculosis: chemotherapy in young adults; analysis of the combined results of three Medical Research Council trials (M. Daniels and A. B. Hill), 1162 (O); 1179 (L)

Stress: *Annual Report on Stress* (H. Selye), 1288
— prolonged, effect on adrenal cortical function, 1055; 1249 (C)
STRONG, G.: Parkinson's syndrome following severe herpes ophthalmicus, 533 (correspondence), 820
STRUMZA, M. V.: Diffusion respiration, 1358
Styes, recurrent, prevention, 1259
Succinylcholine chloride (H. Richards and H. R. Youngman), 1334 (O); 1342 (A)
— — effect on respiration, 440
— iodide (H. Richards and H. R. Youngman), 1334 (O); 1342 (A)
— prolonged respiratory paralysis after administration, 866, 971 (correction, 1084), 1027, 1135, 1342 (A), 1352
— relaxant in electro-shock, 1027
SUGAR, H. S.: *Glaucomas*, 586
Suicide, attempted and successful, 1130
— — responsibilities of medical practitioner, 339, 490, 657, 761, 921
— methods used, 849
Suicides in prison in England and Wales, 391
Sulphamerazine, dangers of, 713, 870
Sulphonamides: Effect on mortality rate in pneumonia, 972
— relation between sensitivity to thiopentone, sulphonamides, and sunlight (F. Evans and J. Gould), 417 (O); 477 (A)
Sulphone treatment of pulmonary tuberculosis, 1224
Sulphur taste and smell in association with cancer, 448
SUMMERS, G. A. C. *See* POOLE, J. C. F., 353
SUMMERS, V. K.: Anaemia of hypopituitarism, 787 (O)
— *See* SHEEHAN, H. L., 1214
SUMMERSKILL, Edith, on prescribing costs (parliamentary note), 772
SUMNER, J. B., and MYRBÄCK, K. (Editors): *Enzymes. Chemistry and Mechanism of Action*, 423
Sunburn, prevention and treatment of, 1403
Sunlight, chronic effects of exposure to, 1404
— clinical effects of exposure to, 1345
Suprarenals. *See* Adrenals
Supraspinatus tendon, rupture of, 41

SURGERY:
Anatomy in Surgery (P. Thorek), 639
Apologie and Treatise of Ambroise Paré, edited by G. Keynes (C. Singer) (Nova et Vetera), 1129
Diabetes mellitus in surgical patients (C. F. Rolland), 737 (O)
Eighth International Congress, 612
Nomenclature of operations, 330, 492, 715
Operating glasses (G. Romanes), 1351
Operative; T-piece technique for general inhalational anaesthesia; safe method of reducing operative bleeding (J. Bullough), 28 (O)
— trapping the surgeon's sweat, 1197

SURGERY (*continued*):
Plastic: *Plastic Surgery: An Introduction for Nurses* (C. R. McLaughlin), 1339
Plastics, 750 (L)
Recommendations for colouring surgical fluids, 982
Surgical boots (parliamentary note), 497
Surgical Travellers' Club, 776
SUTHERLAND, J.: Common cold, 222
SUTHERLAND, J. W. *See* CHAPMAN, T. L., 72
SWAN, H. J. C.: *Noradrenaline, adrenaline, and the human circulation*, 1003 (O)
SWANN, P. G. *See* LOCKET, S., 254
SWANSTON, Catherine: Meaning of occupational health, 1131
Sweat, deficiency, thermogenic, 1346
— glands, neural transmission in, 248
— hat band for catching surgeon's sweat during operations, 1197
Sweats, nocturnal, investigation and treatment
Sweden: Code of medical ethics, 181
SWEET, R. D.: Ethics of leucotomy, 1029
SWEET, R. H.: *Thoracic Surgery*, 424
SWEETNAM, W. P.: Dangers of enemata in megacolon, 866
— MURPHY, E. F., and WOODCOCK, R. C.: Acute idiopathic paroxysmal cold haemoglobinuria of non-syphilitic type in a child, 465 (O)
SWYER, G. I. M.: Male infertility, 218
— Hormone assays in normal and abnormal pregnancies, 619 (O)
SWYNNERTON, B. F., and TRUELOVE, S. C.: Carcinoma of the stomach, 287 (O); 313 (L); (correspondence), 492, 708, 1029, 1301
SYMCOTTS, John: *A Seventeenth Century Doctor and his Patients. John Symcotts*, 1592?–1662 (F. N. L. Poynter and W. J. Bishop), Bedfordshire Historical Record Society Publications, Vol. XXXI (Nova et Vetera) (C. Singer), 1350
SYMES-THOMPSON, Henry Edmund, obituary of, 278
Sympathectomy for Raynaud's disease; results of ganglionectomy and pre-ganglionic section compared (J. B. Kinmonth and G. J. Hadfield), 1377 (O)
— in hypertension, 1247
— in treatment of embolism and thrombosis of the cerebral vessels (R. Leriche), 231 (O); 262 (L); 381 (C), 486 (C), 487 (C), 777
Syncope and fatal wasp stings, 1366
"Synkavit," effect in hypoprothrombinaemia, 414
Syphilis: Kahn tube test with dropping-pipettes (O. Khairat), 582 (O)
— Purpura and pancytopenia complicating arsenotherapy (M. G. Nelson), 300 (O); 714 (C)
Syringe service for a department of anaesthetics (T. H. S. Burns), 1127
Syringes, safety adapter for, 1029

T

Tabes dorsalis; treatment of lightning pains, 1091, 1312
Tachycardia, paroxysmal, in pregnancy, 174
TAIT, E. F.: *Textbook of Refraction*, 1117
TALBOT, Ernest, obituary of, 872
Tampon, vaginal: effect on vaginal flora (Elizabeth N. Brand), 24 (O)
Taste: Fifth and seventh cranial nerves in relation to nervous mechanism of taste sensation; a new approach (W. Harris), 831 (O); 860 (A)
— theory of, 44
Tata Memorial Trust awards, 114, 1363
TATLOW, W. F. T., ARDIS, J. A., and BICKFORD, J. A. R.: *Synopsis of Neurology*, 1010
Tattooing as possible means of transmitting viral hepatitis (A. C. S. Hobson, D. E. Fraser, and N. H. Newman), 1111 (O)
TAYLOR, Frank (Horsham): Incontinentia pigmenti, 439
TAYLOR, Frank (London): Epigastric hernia and dyspepsia, 655
TAYLOR, Frederick William, obituary of, 823
TAYLOR, J. D.: Post-mortem diagnosis of air embolism by radiography, 890 (O)
TAYLOR, Joan: Food-poisoning, 593
TAYLOR, John Frank, obituary of, 1410
TAYLOR, S.: Fouling the nest, 1195
TAYLOR, W. H.: Fantus estimation of urinary chloride, 275
TAYLOR, W. O. G.: Nomenclature of diseases and operations, 492
— What doctors prescribe, 600
Tea, the national drug (parliamentary note), 552
TEARE, D.: Medico-legal aspects of abortion, 915; 911 (A)

TEETH: *See also* Caries, dental; Dentistry
Appeal for post-mortem material, 549
Carious, bacteriology of, 1418
Danger of multiple extraction, 222
Dangers of *x*-ray mirror for dental fluoroscopy, 827
Dental disease among 14-year-old London schoolchildren in 1947 and 1950 (Lady May Mellanby and Helen Mellanby), 7 (O)
N.H.S. charges for dental treatment (parliamentary note), 978, 1034
On edge, 1041
Tooth embedded in tonsil, 766
— in lung; death after 19 years (medico-legal note), 718
Teething powders causing mercury nephrosis, 358; 874 (parliamentary note)
Telephones; party lines for doctors (parliamentary note), 1360
Television; its effect on eyes, 558
— social consequences of, 1017 (A)
— transmission of surgical operations, 1344 (A)
Temperature: Accurate reading of oral temperature, 1418
— normal, 1259
TEMPLE, G. M., Hunterian Society Gold Medal awarded to, 1257
Tenosynovitis and tendovaginitis (D. Ll. Griffiths), 645, (correspondence), 766
TEPPER, R. *See* FRANKLIN, L. M., 485
Terminology. *See* Nomenclature
"Terrabon," 419
"Terramycin" in infections in infants and children (B. Wolman and A. Holzel), 419 (O); 546 (C), 712 (C), 715 (C), 762 (C)
— supplies for general practice, 1023, 1089; 1197 (C)
TERRY, R.: Risks of needle biopsy of the liver, 1102 (O); 1249 (C)
— Erythrocyte sedimentation in anaemia, 1407
Testicles: Orchitis in Bornholm disease (R. J. G. Morrison and J. P. Baird), 198 (O)
Testosterone therapy in endometriosis, 286
Tetanus, cephalic, with report of case (L. Bagratuni), 461 (O)
— neonatorum treated with penicillin and aureomycin; two cases (Mary E. Egerton), 470
Tetany and fatal hypokalaemic alkalosis during liquorice and P.A.S. therapy (N. J. Roussak), 360 (O)
Tetraethylammonium chloride in treatment of disseminated sclerosis, 880
Tetraethylthiuramdisulphide, indications and contraindications in alcoholism, 931
— used for attempted suicide, 849
THACKRAY, A. C. *See* FRIEND, J., 574
THEOBALD, G. W. *See* BENSTEAD, Nancy, 407
Therapeutics: The bottle of medicine and the use of the placebo, 149 (L); 326 (C)
— *Cornell Conferences on Therapy* (Editors: H. Gold and others), 205
THICK, G. C.: Ryle's tube in the trachea, 486
Thicknesse, Philip: *Dr. Viper. The Querulous Life of Philip Thicknesse* (P. Gosse), 1338
Thiosemicarbazone, bone marrow depression due to, 1302
THOMAS, Betty: Ryle's tube in the trachea, 486
THOMAS, D. F.: Surgical treatment of sciatica, 325
THOMAS, J. M., and FEENY, P. J.: Accidental generalized vaccinia, 1249
THOMAS, J. W. Tudor, as B.M.A. President 1953–4, 1181 (A)
THOMAS, M. E. M.: "Epidemic" abdominal colic associated with steatorrhoea, 691 (O)
THOMAS, W. M. *See* WISEMAN, D. C., 141
THOMSON, J., and KEAY, K. R.: Case of acute beriberi complicating polyserositis in Britain, 295 (O)
THOMSON, M. L. *See* WILSON, Vera K., 358, 484
THOMSON, W. O.: Naevi in pregnancy, 162
Thorax. *See* Chest
THOREK, P.: *Anatomy in Surgery*, 639
THORNTON, George Lestock, obituary of, 110, 332
THORPE, F. T.: Post-leucotomy anxiety, 49
Throat, streptococcal infection of, 969
Thromboangiitis obliterans, causal relationship to trauma, 1143
Thrombosis and embolism of cerebral vessels; treatment (R. Leriche), 231 (O); 262 (L); 381 (C), 486 (C), 487 (C), 777
— cerebral, anticoagulant therapy contraindicated, 58
— coronary, cholesterol tolerance in (I. Wang), 1278
— prolonged anticoagulant therapy with heparin (A. Wynn, J. F. Goodwin, and Angela Birbeck), 893 (O)
Thumb: "Trigger-thumb" simulating paralysis; report of two cases (A. Zinovieff), 199 (O)
THWAITES, J.: Doctors' opinion in lay press, 599
— Attempted suicide, 657
Thyroid: Changes in basal metabolism, serum-protein-bound iodine, and cholesterol during treatment of hypothyroidism with oral thyroid and L-thyroxine sodium (J. D. Robertson and H. F. W. Kirkpatrick), 624 (O)
— Chilblains and hypothyroidism, 711
— hormone, level in pregnancy, 621
Thyrotoxicosis: *Thyreotoxikosen und Antithyreoidale Substanzen* (H. W. Bansi), 1339
— treatment, 49
L-Thyroxine sodium treatment of hypothyroidism, changes in basal metabolism, serum-protein-bound iodine, and cholesterol during (J. D. Robertson and H. F. W. Kirkpatrick), 624 (O)
TILLMAN, W. G.: Mepacrine in skin disease, 1406
Tinea capitis (*M. audouini*) in residential school (A. L. Walby), 1114 (O)
Tissue reactions to protein sensitization (J. R. Squire), 1 (O); 35 (L)
— Traumatic autografting, 274
Titmuss, R. M., on the behaviour of consultants towards hospital patients, 1195, 1244
Tiverton and District Hospital centenary, 1325

TOBACCO:
Arsenic in cigarettes (parliamentary note), 824
Pills for prevention of smoking, 118, 558
Smoking and cancer of lung (parliamentary note), 335
— medical aspects (R. B. Scott), 671 (O); (correspondence), 869, 975

TOBACCO (*continued*):
Tobacco and the Cardiovascular System. The Effects of Smoking and of Nicotine on Normal Persons (Grace M. Roth), 803
Value of cigarettes after operation, 1354
α-Tocopherol in diabetic gangrene, 276 (correction, 493)
— vitamin E in treatment of Dupuytren's contracture (H. J. Richards), 1328 (O)
TODD, J.: Hypnotism in medicine, 922
TODD, R. McL.: Cost of treatment, 546
TONDURY, G.: *Angewandte und topographische Anatomie*, 696
Toluene, toxic effects, 174
TOMSKI, H. W.: What doctors prescribe, 436
Tongue, geographical, causes and treatment, 448, 670
Tonsil, tooth embedded in, 766
Tonsillitis, effect of "terramycin" in, 419
Tonsils and adenoids; dogmas and doubts, 722
— — — fatal staphylococcal septicaemia following removal (E. J. G. Wallace and O. G. Jones), 1231
TOPPING, A., address at Royal Sanitary Institute Health Congress, 1021; 1196 (C), 1246 (C)
— elected President of Society of Medical Officers of Health, 1142
Tourniquet, forgotten (medico-legal note), 225, (correspondence), 328, 437, 605
TOWN, A. E.: *Ophthalmology*, 1117
Towns: *The Making of our Towns* (Sir William Savage), 1177
TOWNSEND, E.: Mr. Bevan, 870
Toxaemia, fatal, after blood transfusion (medico-legal note), 1036
"Toxaphene," 543

TOXICOLOGY:
"Anthisan" poisoning, treatment of, 163
Blood cholinesterase level in diagnosis of insecticide poisoning (W. N. Aldridge and D. R. Davies), 945 (O); 1015 (L); (correspondence), 1135
Ethyl acetate, toxic effects, 174
Mercury nephrosis in young children, with special reference to teething powders containing mercury (Vera K. Wilson, M. L. Thomson, and A. Holzel), 358 (O); 370 (A); (correspondence), 484, 931
— poisoning, chronic, in latent finger-print development (G. Forbes and J. White), 899 (O); (correspondence), 1131, 1406
Methyl bromide poisoning, 1122 (A)
Poison antidotes on labels (parliamentary note), 1413
Toluene, toxic effects, 174
Toxins, bacterial food-poisoning due to, 593
Trachea: Aid to analgesia of the larynx and trachea (T. Dinsdale) (preparations and appliances), 707
— stomach tube in, 382, 486, 710, 715, 920
Trachoma, treatment; conclusions of W.H.O. Expert Committee, 721
TREECK, A. S. van. *See* EICKEN, C. von, 1072
TRENCHARD, H. J., and GRENVILLE-MATHERS, R.: Tuberculosis incidence in children, 1194
Trichiniasis in Barry (vital statistics), 1363
Trichlorethylene analgesia in labour, 108
— decomposition of, 1405
— not to be used by midwives, 280
Triethylene melamine in Hodgkin's disease and lymphosarcoma, 1343 (A)
Trigger-finger and thumb, 647
"Trilene." *See* Trichlorethylene
"Tromexan"-induced hypoprothrombinaemia, effect of vitamin-K preparations on, 412
Tropical disease: Facilities for treatment in Britain (parliamentary note), 1360
— fatigue, 815
— medicine: Hygiene for the medical tramp, 1365
— medicine: Ross Institute Industrial Advisory Committee, 479 (A)
— medicine: Tolerance to hot climate, 1328
— ulcers, treatment of, 493, 764
TRUELOVE, S. C. *See* SWYNNERTON, B. F., 287
TRUMAN, S. R.: *The Doctor. His Career. His Business. His Human Relations*, 586
Trypsin estimation in duodenal juice (I. Gordon, B. Levin, and T. P. Whitehead), 463 (O)
TSONGAS, A. G.: *Nutrition Work in Greece*, 145; 384 (C)
"Tubarine," disadvantages in electric convulsion therapy, 109
Tuberculin: Depression of tuberculin sensitivity in guinea-pigs by certain antituberculous surface-active agents (P. D. Hart, D. A. Long, and R. J. W. Rees), 680 (O); 702 (A)
— post-vaccination test, 840
— reaction to, 4
— Survey Committee, Medical Research Council, 1397 (L)

TUBERCULOSIS:
Atypical abdominal tuberculosis with haematological manifestations (J. Friend and A. C. Thackray), 574 (O)
B.C.G. vaccination, 270
— — efficacy of, 147 (L)
— — report on mass campaign in Malta, 1950 (V. Zammit-Tabona), 837 (O)
— vaccine, risks in handling, 1143
Beds in sanatoria; spacing and construction, 1418
Breast-feeding: indications and contra-indications, 879
Cod-liver oil for patients, 1254
Conference of International Union against Tuberculosis, 284

TUBERCULOSIS (*continued*):
Contracted by nurse; industrial injury benefit awarded (medico-legal note), 443
Education Institute; clinical courses, 228
Employment of tuberculous patients, 885
In families with B.C.G.-vaccinated and non-vaccinated children (A. W. Dickie), 682 (O); 1194 (C)
In foreign workers (parliamentary note), 1412
In industry, 1012 (L)
In mental hospitals (B. T. Mann), 1228 (O)
In Singapore (parliamentary note), 1413
In Tyneside (parliamentary note), 609
Joint Tuberculosis Council, joint annual report, 7; annual luncheon, 1203
Knee-joint; treatment with intra-articular streptomycin (R. T. Ahern and G. P. Arden), 466 (O); (correspondence), 766, 974, 1194
Modern Practice in Tuberculosis (Editors: T. H. Sellors and J. L. Livingstone) (in 2 vols.), 747
N.A.P.T. clinical conference, 929
Notification regulations (vital statistics), 981
Pelvic tuberculosis treated with *iso*nicotinic hydrazide, 1028
Pulmonary, and pregnancy (J. R. Edge), 845 (O); 862 (A); (correspondence), 1030
— chemotherapy in young adults; analysis of the combined results of three Medical Research Council trials (M. Daniels and A. B. Hill), 1162 (O); 1179 (L)
— lung resection for, 34 (L)
— prevention of streptomycin resistance by combined chemotherapy; M.R.C. investigation, 1157 (O); 1179 (L); (correspondence), 1302
— sleeping posture in, 316 (A)
— treatment with sulphone and streptomycin (P. W. Edwards, A. C. Penman, and L. J. Cutbill), 1224 (O)
Rebel patients, 1299
Sanatoria waiting-lists (parliamentary note), 497
Scotland (parliamentary note), 334
— mortality in 1949 (vital statistics), 499
Survey in school-children in the Calder Valley (S. Keidan and others), 1390 (O); 1397 (L)
Treatment at Danish sanatoria, 634
— by "pendulum swinging" (medico-legal note), 665
— calciferol as adjuvant, 986
— dihydrostreptomycin contraindicated, 1076 (A)
— with nicotinic acid derivatives, 478, 858 (L), 983, 1015 (A); (correspondence), 868, 970, 1028, 1081, 1133; Ministry of Health statement, 775
Tuberculin; post-vaccination test, 840
— reaction to, 4
— Survey Committee, M.R.C., 1397 (L)
— sensitivity; depression in guinea-pigs by certain antituberculous surface-active agents (P. D. Hart, D. A. Long, and R. J. W. Rees), 680 (O). 702 (A)
Tuberculous glands in the neck, treatment of, 285, 830
— meningitis, treatment with streptomycin and *para*-aminosalicylic acid (S. R. Jamieson), 83 (O) (corrections, 165, 223); 329 (C)
— pleural effusion, primary, in older age groups (R. F. Robertson), 133 (O)
Vitamin supplements in, 986
Tubocurarines compared, 1144
TUCKMAN, E. *See* FRANKLIN, L. M., 485
TUDOR-HART, J.: Economics and doctors, 818
TULLOH, H. P.: Negative benzodioxane test in presence of phaeochromocytoma, 531 (O)
Tumours: Treatment with bacterial products, 429 (A)
TURNER, P. P.: Medical aspects of tobacco-smoking, 975
TUTHILL, J. F.: Stage hypnotism, 816
TWEED, J. M. *See* COPEMAN, W. S. C., 397
Tyneside: Tuberculosis (parliamentary note), 609
Typhoid fever vaccination, 270
— *see also* Enteric fever

U

UCKO, H.: Treatment of hypertension, 1354
Uganda: Annual clinical conference of Uganda Branch of B.M.A., 158; 820 (C)
ULCER: *For* Duodenal ulcer, Gastric ulcer, Peptic ulcer, *see under* Peptic ulcer
Granulomatous, of nose treated with chloramphenicol (I. B. Sneddon and J. Colquhoun), 298 (O); 491 (C)
trophic, after infantile paralysis, treatment of, 1312
tropical, treatment, 220, 493, 794
"Ulisse" Prize for 1952, 612
Ulster Paediatric Society, 612, 1309
Ultra-violet light, skin resistance to, 162
Umbilicus, bleeding from, causes and treatment, 1092
Unesco: *The Haiti Pilot Project. Phase One*, 1947–49, 96 (A)
UNGAR, J., and MUGGLETON, P. W.: Accumulation of diethylaminoethanol ester of penicillin in inflamed lung tissue, 1211 (O); 1238 (A)
— *See* SORSBY, A., 119
UNGLEY, C. C.: Therapeutic value of vitamin B_{12}, 161

UNITED STATES OF AMERICA:
Cost of medical treatment, 1176
Interns in Army hospitals, 953
Medical student intake, 187

UNITED STATES OF AMERICA (*continued*):
Rheumatic diseases (J. J. R. Duthie), 341 (O); 602 (C)
Study grants, 1364

UNIVERSITIES:
Aberdeen: Appointments, 391
Degrees conferred, 112
Belfast, Queen's University: Appointments, 1307
Bull, G. M., appointed Professor of Medicine, 1307
Degrees conferred, 112
Pritchard, J. J., appointed Professor of Anatomy, 1307
Birmingham: Appointments, 281, 719, 1255, 1413
Degrees conferred, 281
Honour schools in medical sciences, 1413
Leonard Parsons Memorial Lectureship, 281
Skin research unit, 1255
Bristol: Examination results, 53, 1036
British Columbia: Paterson, D. H., appointed Clinical Professor of Paediatrics, 225
Cambridge: Appointments, 52, 168, 719, 1306, 1361
Copeman Medal, 497
Degrees conferred, 52, 336, 497, 554, 825, 1201, 1254, 1361
Examination results, 111
Honorary degrees conferred, 1306
Matthews, B. H. C., elected Professor of Physiology, 280
Raymond Horton-Smith Prize, 52
Research fellowships, 336
Dublin: Appointments, 825
Lecturers, 825
School of Physic, Trinity College: Awards, 1036
Degrees conferred, 112
Examination results, 1036
Durham: Degrees conferred, 53
Edinburgh: Appointments, 1201
Degrees conferred, 52
Diplomas conferred, 52
Edinburgh University Club of London, 1415
Glasgow: Degrees conferred, 280, 825, 1036
Leeds: Appointments, 497
Degrees conferred, 1255
Donations, 497
J. W. Wootton Fellowship, 1255
Oakley, C. L., appointed to Brotherton Chair of Bacteriology, 1255
Stewart retirement fund, 1255
Liverpool: Examination results, 53, 825
London: Appointments, 53, 281, 554, 825, 1254
Barwell, C. F., appointed to Goldsmiths' Company's Chair of Bacteriology at London Hospital Medical College, 1361
Constitution of Senate (parliamentary note), 1199
Conway Evans Prizes, 979
Degrees awarded, 281
Diplomas awarded, 53, 719, 1254
Examination results, 112, 1255, 1306
Garnham, C. C., appointed to the Chair of Medical Protozoology, London School of Hygiene and Tropical Medicine, 1254
King's College Hospital Medical School: Wiltshire Memorial Research Scholarship, 1307
Morgan, W. T. J., appointed Professor of Biochemistry, 336
Scholarships, 1036
School of Pharmacy granted Royal Charter of Incorporation, 1363
Teachers recognized, 554, 1255
William Julius Mickle Fellowship, 554
Manchester: Appointments, 391
Examination results, 53, 825
Honorary degrees conferred, 825
Radden, H. G., appointed Professor of Dental Surgery, 1307
Oxford: Appointments, 111, 280, 719, 1036, 1254, 1306
Corner, G. W., appointed George Eastman visiting professor, 111
Degrees conferred, 280, 336, 719, 825, 1201
Honorary fellows, 497
Intake of medical students, 455
Scholarships, 280
St. Andrews: Appointments, 280
Examination results, 280
Honorary degrees conferred, 497
Sheffield: Appointments and retirements, 554, 825, 1255
Degrees conferred, 112
Wales: Diplomas granted, 281
Welsh National School of Medicine, 281
University College Hospital: Metabolic and radio-isotope work, 648, 667
University Grants Committee: Medical teaching staff without experience in private practice recommended, 974
— — — *University Development; Interim Report on the Years* 1947 *to* 1951, 479 (A) (correction, 614)
University of Durham Medical Gazette, 1257
University of Leeds Medical Journal, 611
Urethra: Analgesia for instrumentation, 395
Urinary system: Bladder paralysis; a peculiar syndrome in natives of South Central Africa (M. Gelfand and E. E. Barnett), 1172 (O); 1408 (C)

URINE:
Chloride estimation, 275
— — by Fantus method, 48

URINE (*continued*):
Frequency of micturition; causes and treatment, 372
High ketosteroid level, significance of, 879
Incontinence in a woman, 110, 277
Stress incontinence; preliminary report on new method of treatment (G. G. Lennon), 1384 (O)
Urography: *Clinical Urography: An Atlas and Textbook of Roentgenologic Diagnosis* (W. F. Braasch and J. L. Emmett), 1011
Urology: *Clinical Pediatric Urology* (M. Campbell), 1071
— *Urology of Childhood* (T. T. Higgins, D. I. Williams, and D. F. E. Nash), 695
Urticaria, intractable, treatment of, 1417
— neonatal, 1134
U.S.S.R.: Invitation of Russian doctors to Britain proposed, 50, 165, 548, 652, 710, 818, 1257
— *Soviet Genetics* (A. G. Morton), 472; 708 (C)
— *Soviet Psychiatry* (J. Wortis), 473
UTERUS:
Cervix, erotic sensitivity of, 273, 437, 656
— involution of (H. C. McLaren), 347 (O); (correspondence), 655
— post-natal erosions, 349
— post-natal histology, 349
— sensation in, 273, 437, 656
Intra-partum rupture (R. Hodkinson), 308; 438 (C), 604 (C), 973 (C)
Post-natal mucous plug, 348

V

Vaccination. *See also* Smallpox
— for smallpox, fatal encephalomyelitis following (vital statistics), 1037
— reactions to, 483
Vaccines, reactions to, 213
Vaccinia, generalized, accidental; case (O. A. Finn, J. C. Dick, and J. S. Stevenson), 1067 (O); (correspondence), 1134, 1249
Vagina: Bacteriology of flora after use of internal tampons (Elizabeth N. Brand), 24 (O)
— smear, post-natal, 349
VALDEZ, F. N.: Male infertility, 106, 328
VALLERY-RADOT, P.: *Études Cliniques, Expérimentales et Thérapeutiques sur l'Allergie*, 204
Valvotomy for mitral stenosis; further report, on 100 cases (C. Baker and others), 1043 (O); 1073 (L)
VANDEPITTE, J. M.: Sickle-cell anaemia in Africa, 920
Vanishing creams incorporating skin medicaments, 670
VAN PELT, S.: Hypnotism in medicine, 487
— Stage hypnotism, 921
Varicella. *See* Chicken-pox
Varicose Veins and their Complications, Treatment of (S. Rivlin), 260
Variola. *See* Smallpox
VARTAN, K.: Family planning, 766
VAUGHAN, Kathleen: *Exercises Before Childbirth*, 1118
VAUGHAN-MORGAN, Josephine. *See* GREENE, R., 17
VEDDER, Edward Bright, obituary of, 441
Venereal disease: *See also* Gonorrhoea; Syphilis
— *Venereal Diseases described for Nurses* (R. C. L. Batchelor and Marjorie Murrell), 365
Venesection, indications for, 1260
"Veriloid," hypotensive action of, 598
Vertigo, aural, value of histamine skin test in, 869
VETTEN, K. B. *See* DEVITT, D. C., 1303
VICKERS, A.: Strained eyes, 502
VICKERS, H. E.: Experiment in peacemaking, 710
Victoria Medical Foundation, 929
Victorian pennies appeal, 1309
VILVANDRE, G. E.: Carcinoma of the stomach, 708
Vinci, Leonardo da. *See* Leonardo da Vinci
Vision: *Textbook of Refraction* (E. F. Tait), 1117
— of drivers; relation to road accidents, 108
VITAL STATISTICS:
Census: Changes of population in 1951 Census, 226, 283
Cheltenham; puzzling dermatological affection, 444, 1203; 1303 (C)
Demographic revolution, 1289 (L)
Diptheria, 774
— at Darwen, Lancs, 826
— graphs, 170, 775, 827, 876
— immunization in 1950, 499
Dysentery; graphs, 54, 114, 171, 447, 498, 553, 610, 928, 1308, 1363, 1415
England and Wales: Registrar-General's quarterly returns, 982
Enteric fever: graphs, 170, 666, 721, 981, 1038, 1089, 1141, 1202, 1256, 1309, 1362, 1414
Industrial accidents and diseases, 54, 337, 827, 981, 1256
Infant mortality rate, 169, 282, 982
Infectious diseases; graphs, 54, 114, 170, 226, 282, 336, 392, 445, 498, 553, 610, 666, 721, 774, 775, 827, 876, 928, 981, 1038, 1088, 1140, 1202, 1308, 1362, 1363, 1414–5
— — incidence in 1951, 169
— — notifications, 54, 113, 169, 227, 282, 336, 392, 444, 498, 553, 610, 666, 721, 775, 826, 876, 928, 981, 1037, 1089, 1140, 1202, 1256, 1308, 1362, 1414
VITAL STATISTICS (*continued*):
Influenza; graphs, 171
— outbreak in Glasgow, 720
Ireland: Registrar-General's quarterly returns, 226, 1256
— Report of Department of Health for 1950–1, 1255
London health in 1951, 113
Measles; graphs, 170, 445, 499, 553, 610
Meningococcal infection: graphs, 170, 282, 336, 392, 981, 1038, 1088, 1141, 1202, 1256, 1414
Ophthalmia neonatorum; graphs, 170, 981, 1038, 1088, 1141, 1203, 1256
Pneumonia; graphs, 54, 114, 170, 226, 282, 336, 392, 445, 499, 553, 610, 666, 721, 774, 827, 876, 928
Poliomyelitis, acute, and polioencephalitis; graphs, 170, 981, 1038, 1088, 1140, 1203, 1256, 1308, 1362, 1415; notifications, 1255
Population: Distribution changes shown by 1951 Census, 283
Puerperal pyrexia and puerperal fever: graphs, 171
Road accidents, 282, 499, 721, 1363
Salmonella minnesota infection, 53
Scarlet fever; graphs, 54, 114, 171, 226, 282, 336, 392, 666, 721, 775, 827, 876, 928
Scotland: Causes of death in 1949, 337
— Registrar-General's quarterly return, 875
Singapore; annual report of Medical Department, 114
Smallpox; death following vaccination, 1037
— in Hampshire, 775
— in Lancashire, 444, 498, 478 (A), 553, 610, 666, 720, 775, 875, 928, 981, 1038, 1088
— in Mexico, 1088
— in Swansea, 981
— notifications, 826
Trichiniasis in Barry, 1363
Tuberculosis; mortality in Scotland in 1949, 499
— notification regulations, 981
Weekly summary: 55, 113, 171, 226, 281, 337, 391, 444, 498, 554, 611, 667, 720, 774, 826, 877, 927, 980, 1037, 1088, 1140, 1202, 1257, 1308, 1362, 1414
Whooping-cough; graphs, 54, 114, 170, 227, 282, 337, 392, 445, 499, 553, 610, 666, 721, 774, 827, 876, 928, 1309, 1363
VITAMINS: *See also* Food; Nutrition
A, poisoning due to, 539 (A)
B_{12}, 640 (L); 1357 (C)
— in megaloblastic anaemia, 220, 764
— influence on growth, 1388; 1401 (A)
— Is it a tonic? 1041
— isolation, chemistry, and therapeutic value, 160
— side-effects of a preparation of (P. D. Bedford), 690 (O)
E, in treatment of Dupuytren's contracture (H. J. Richards), 1328 (O)
— α-Tocopherol in diabetic gangrene, 276 (correction, 493)
Effect of agene on, 614
Fat-soluble, for children, 1144
In breast milk, 229
K: Effect on hypoprothrombinaemia induced by dicoumarol and "tromexan" (A. S. Douglas and A. Brown), 412 (O); 603 (C), 763 (C)
— in treatment of acute hepatitis (H. Mossberg), 1382 (O)
Vitamin supplements in tuberculosis, 986
Vitamins. A Digest of Current Knowledge (L. J. Harris), 310
VOGT, Marthe L., elected, F.R.S., 703
Vomiting during anaesthesia, 711
Vulva, atresia of, causing urinary incontinence, 110
— periodic oedema of, 1092

W

WADDY, F. F.: Cost of T-piece anaesthetic technique, 384, 654
WADDY, S. H.: Family planning, 867
— Period of gestation, 1132
WAKELEY, Sir Cecil: Clinical research in the National Health Service, 917; 974 (C), 1030 (C), 1196 (C)
— Corneal grafts, 544
— baronetcy conferred on, 1299
WALBY, A. L.: Tinea capitis (*M. audouini*) in residential school, 1114 (O)
WALES, Edward Garneys, obituary of, 1305
Wales, University of. *See* Universities: Wales
WALK, A.: Discharge of certified mental patients, 1193
WALKER, Cynthia M.: Testing for serratus anterior weakness, 1393
WALKER, K.: *Marriage: A Book for the Married and about to be Married*, 1178
WALKER, W.: Haemolytic disease of the newborn, 108
Walking, re-education in, 1300
WALKOWSKI, J. *See* GALPINE, J. F., 1069
WALLACE, C. P.: Mr. Bevan, 1030
WALLACE, Sir David, obituary of, 976
WALLACE, E. J. G., and JONES, O. G.: Fatal staphylococcal septicaemia following removal of tonsils and adenoids, 1231
WALLIS, H. R. E.: Funnel chest, 715
WALLIS, Preston Richard, obituary of, 442
WALLS, N. S.: Anaesthetic explosions, 820
WALTON, A. M. *See* MONCRIEF, A., 43
WALTON, J. N., and LILL, Norma D.: Bleeding Meckel's diverticulum, report of two cases, 88 (O); (correspondence), 221
WALTON, Sir James: Basic principles of atomic structure, 99
WALTON, L. H. F.: Sister Kenny, 1082
WANG, I.: Cholesterol tolerance in coronary thrombosis, 1278 (O)
WAPSHAW, H.: Repair of damaged accessory hepatic duct by anastomosis with cystic duct, 906
War: Medical Association for the Prevention of War; first national conference, 1089
WARD, C. W.: Dermatitis after using "pyrotenex," 905; 1249 (C)
WARD, J. M.: Wandering spleen, 1407
WARD, T.: Exchange transfusion in severe anaemia, 631 (O)
WARDLE, E. N.: Primary generalized arthritis, 383
WARTENBERG, R.: Head-dropping test, 687 (O)
Warts of the feet; diagnosis and treatment (L. Forman), 480; 712 (C)
Wasp stings, fatal syncope due to, 1366
— — treatment with calcium salt, 340
Watches: People on whom watches will not keep time, 1366
Water: Cleaning of fresh-water tanks and filters, 292
— softeners, effect on lead pipes, 1417
— sterilization in Armed Forces, 532
— storage of drinking-water, 724
— supply of London in wartime, 539 (A)
WATSON, A.: Childhood factors in duodenal ulcer, 109
WATSON, D. *See* KERSLEY, G. D., 438, 603
WATSON, G. M., and WITTS, L. J.: Intestinal macrocytic anaemia, 13 (O)
WATSON, T. A., and BERESFORD, O. D.: Educating the public about cancer, 107
WATSON-WILLIAMS, E.: Is Latin dead? 1197
WATTS, C. A. H.: Discharge of certified mental patients, 1193
WAUGH, W. G.: Cortisone in rheumatism, 602
Wax baths, 1417
WEBBER, B.: Mercury in paediatrics, 484
WEBER, F. P.: Proctalgia fugax, 710
WEBSTER, D. D., and BLADES, A. N.: Islet-cell tumour of pancreas, 307 (O)
WEBSTER, Jean M.: Acute infectious lymphocytosis, 324
Weed-killers: Agriculture (Poisonous Substances) Bill (parliamentary notes), 1035, 1199, 1252, 1360
— poisoning due to; blood tests for agricultural workers, 1014 (L); 1024; 1135 (C)
Weights and measures: Metric system, 819
WEIR, J. B. de V. *See* DURNIN, J. V. G. A., 1006
WEISS, T. E. *See* DERBES, E. J., 1072
WEITZNER, H. A.: Gout and insulin hypoglycaemia, 1251
WELCH, H., and LEWIS, C. N.: *Antibiotic Therapy*, 1339
WELCH, J. D.: Inefficiency in the out-patient department, 1022
Welding and health, 1239 (A)
WELLISCH, E.: Jocasta complex in adoptive mother, 817
West African Medical Journal, 877
West Indian Medical Journal, 152 (A)
West Indies: Appendicitis; incidence, aetiology, treatment and prognosis (A. Leacock), 1347
West Middlesex Hospital League of Friends, 422
WESTLAKE, E. K.: Congenital indifference to pain, 144
Westminster Hospital; Old Students' Dinner, 1309
WHARTON, John, obituary of, 1136
WHAYNE, T. F. *See* SIMMONS, J. S., 1010
WHEATLEY, D.: Complications of arsenotherapy, 714
— Relief of acute left ventricular failure by "priscol," 1174 (O)
WHEATON, C. E. W.: Leukaemia with primary gynaecological manifestations, 30
WHITBY, Sir Lionel: Advent of the sulphonamides in treatment of pneumonia, 156
— *Medical Bacteriology*, 5th ed., 1072
WHITE, Gilbert Benjamin Mower, obituary of, 1086
WHITE, J. *See* FORBES, G., 899
WHITE, L. L. R. *See* BODIAN, M., 77
WHITE, Margaret Moore: Tubo-uterine implantation, 1355
WHITE, R. H. R.: Acute suppurative parotitis in newborn, 1232
WHITEHEAD, T. P. *See* GORDON, I., 463
WHITEHOUSE, C. S.: Chicken-pox repeated, 710
WHITTY, C. W. M.: Debauchery of honest words, 762
— *See* MARSHALL, J., 847
Whooping-cough, asthmatic attack due to, 1041
— graphs (vital statistics), 54, 114, 170, 227, 282, 337, 392, 445, 499, 553, 610, 666, 721, 774, 827, 876, 928, 1309, 1363
— immunization, 269, 714
WIBAUT, J. P.: *Organic Chemistry*, 1234
WICKES, I. G.: Homes for incurable children, 277
WIENER, A. S.: Haemolytic disease of the newborn, 108
WIGGLESWORTH, R.: Value of organic manures, 1357
WIGLEY, Diana M.: Significance of marrow changes after A.C.T.H., 1135
Wigs issued, 391
WILLCOX, D. R. C.: Haemolytic anaemia and reticuloses, 1322 (O)
William Julius Mickle Fellowship, 554
WILLIAMS, B.: Thumping the chest, 438
WILLIAMS, D. I: Boy or girl? 614
— Diagnosis of intersex, 1264 (O); 1292 (A)
— *See* HIGGINS, T. T., 695

WILLIAMS, D. O., and MCLEAN, E. K.: Treatment of recent transcervical fractures of femur, 580 (O); 592 (A); (correspondence), 764
WILLIAMS, H.: Medical treatment abroad, 1081
— *Between Life and Death*, 696
WILLIAMS, R. E. O.: Streptococcal sore throat, 969
WILLIAMS, W. O.: Gastric carcinoma and acute perforation, 164
WILLIAMSON, G. S.: Peckham Health Centre, 490
Wills, 56, 172, 555
WILSON, Alexander Garrick, obituary of, 51
WILSON, Eleanor J.: A case of ectopia cordis surviving nine hours, 1175; 1358 (C)
WILSON, T., MUNRO, D. S., and RICHARD, D. R.: Proguanil-resistance in Malayan strains of plasmodium vivax, 564 (O); 590 (A)
WILSON, Vera K., THOMSON, M. I., and HOLZEL, A.: Mercury nephrosis in young children, with special reference to teething powders containing mercury, 358 (O); 370 (A); (correspondence), 484, 931
— *See* HOLZEL, A., 194
WILSON, W. R.: Oesophagitis, 1250
WILTHEW, G. A. *See* KEIDAN, S., 1390
Wiltshire Memorial Research Scholarship, 1307
WINSBURY-WHITE, H. P.: Micturition, frequency of, 372; 547 (C)
WINSLOW, C. E. A.: *The Cost of Sickness and the Price of Health*, 642
WISEMAN, D. C., and THOMAS, W. M.: Ovarian denervation for ovarian dysmenorrhoea; review of 70 cases, 141 (O); (correspondence), 272
WITHERS, J. W.: Painful shoulder, 40
Witnesses at Court, mental condition of, 1254
WITTS, L. J. *See* WATSON, G. M., 13
WODDIS, G. M.: More liaison needed, 1407
WOLF, S. *See* GRACE, W. J., 959
WOLFE, G. S.: Second attack of chicken-pox, 1030
WOLFF, E.: Retrolental fibroplasia, 924
WOLFF, H. G. *See* GRACE, W. J., 959
WOLMAN, B., and HOLZEL, A.: "Terramycin" in infections in infants and children, 419 (O); 546 (C), 548 (C), 712 (C), 715 (C), 762 (C)
Women, sexual disorders in, 104, 273, 437, 656, 1135, 1247
WOOD, P. *See* BAKER, C., 1043
WOODCOCK, R. C. *See* SWEETNAM, W. P., 465
WOODS, D. D., elected F.R.S., 703
WOODSIDE, Moya. *See* SLATER, E., 91
WOOD-SMITH, F. G.: Post-operative brachial plexus paralysis, 1115
Woollen fabrics, sterilization (H. F. Barnard), 21 (O); 601 (C)
WOOLLEY, E. J. S.: Schönlein-Henoch (anaphylactoid) purpura treated with A.C.T.H., 259
WOOLMAN, A. M.: Fatal wasp stings, 340
Wootton Fellowship, University of Leeds, 1255
Word-blindness, 36 (A); 325

WORLD HEALTH ORGANIZATION:
Deputy director for European regional office, 544
Discussion on birth control at annual assembly, 1157, 1405
Expert Committee on Trachoma, 721
Fifth Assembly, 1204, 1141
International Pharmacopoeia Committee, 938
Travelling study group of senior public health administrators, 1026
Work of W.H.O., 913

World Veterans Federation, 667
WORTIS, J.: *Soviet Psychiatry*, 473
Wound healing: Emergency operation on patient under A.C.T.H. and cortisone therapy (A. Beck), 636; 1303 (C)
WRAITH, D. G. *See* HART, F. D., 1273
WRIGHT, A. D.: Trapping the surgeon's sweat, 1197
Wright, Sir Almroth, memorial to, 1039
WRIGHT, G. A., and OWEN, T. K.: Poliomyelitis in mother and newborn infant, 800 (O)
WRIGHT, H. B.: Pregnancy tests, 724
WYANT, G. M.: Explosion during anaesthetic, 870
WYNN, A., GOODWIN, J. F., and BIRBECK, Angela: Prolonged anticoagulant therapy with heparin, 893 (O)

X

Xanthomatoses (A. L. Froehlich), 695
X rays. *See* Radiology; Radiotherapy
Xylocaine, 211 (A); 328, 488, 714, 1083 (correction, 1135)
— injection, fatal (medico-legal note), 280; 714 (C)

Y

Yaws as cause of repeated abortion, 830
YELL, W. L., and NEVILLE, N. A.: Limitations of chemotherapy in tuberculosis, 1302
Yellow fever vaccination, 271
YOFFEY, J. M. *See* HUDSON, G., 999
YOUMANS, J. B. (Editor): *Medicine of the Year*, 1951 edition, 205
YOUNG, David Hunter Dow, obituary of, 871
YOUNG, J. H.: Generalized osteoarthritis, 765
YOUNG, M. L.: Sexual disorders in women, 105
— Frequency of micturition, 547
YOUNGMAN, H. R. *See* RICHARDS, H., 1334
Your Health, 1203
YUDKIN, J.: Effect of liver supplement on growth of children, 1388 (O); 1401 (A)

Z

ZAMMIT-TABONA, V.: Mass B.C.G. vaccination in Malta; report on the B.C.G. vaccination campaign in 1950, 837 (O)
ZERNY, C. J.: Treatment of tuberculous meningitis, 329
ZIMMERLI, E.: Physical characters associated with schizophrenia, 932
ZINNEMANN, K. *See* DAWSON, Betty, 740
ZINOVIEFF, A.: "Trigger-thumb" simulating paralysis; report of two cases, 199 (O)
ZUNDEL, W. E.: Cough fracture, 220

KEY TO DATES AND PAGES

The following table gives a key to the dates of issue and the page numbers of the BRITISH MEDICAL JOURNAL and SUPPLEMENT in the first volume for 1952.

Serial No.	Date of Issue	Journal Pages	Supplement Pages
4748	Jan. 5	1– 58	1– 8
4749	„ 12	59– 118	9– 16
4750	„ 19	119– 174	17– 26
4751	„ 26	175– 230	27– 34
4752	Feb. 2	231– 286	35– 46
4753	„ 9	287– 340	47– 58
4754	„ 16	341– 396	59– 64
4755	„ 23	397– 448	65– 72
4756	Mar. 1	449– 502	73– 84
4757	„ 8	503– 558	85– 94
4758	„ 15	559– 614	95–106
4759	„ 22	615– 670	107–112
4760	„ 29	671– 724	113–138
4761	Apr. 5	725– 778	139–146
4762	„ 12	779– 830	147–154
4763	„ 19	831– 880	155–202
4764	„ 26	881– 932	203–208
4765	May 3	933– 986	209–230
4766	„ 10	987–1042	231–240
4767	„ 17	1043–1092	241–246
4768	„ 24	1093–1144	247–254
4769	„ 31	1145–1206	255–280
4770	June 7	1207–1260	281–288
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